The
Southern Surgeon

Subscription in the United States, $4.00

VOLUME V AUGUST, 1936 NUMBER 4

PASSIVE VASCULAR EXERCISES IN THE TREATMENT OF OBLITERATIVE VASCULAR DISEASE

O. P. BOARD, M. D.
Birmingham

UNTIL recent years peripheral vascular disease has attracted little attention from scientific investigators. By reason of this fact the attitude of the profession in this field of endeavor has been one of casualness and indifference. The inevitable results of this affliction have been accepted in a spirit of hopelessness. Medical aid has assumed the line of watchful waiting until obliterative changes forced amputative surgical relief.

In a brief span of five years, beginning about 1930, the revolutionary approach to this subject has arrested the attention of the profession. Among the workers in this newer concept of vascular disease, Brown[1], Morton and Scott[2], Allen[3], Adson[4], Silbert[5], Herrmann and Reid[6], Flothow[7], Landis and Gibbon[8], have been outstanding. Their efforts have stimulated renewed interest, research, and instituted rational and effective methods for the care of these cases, and have presented a working classification.

In our study of peripheral vascular disease the following classification from the Vascular Disease Clinic of the Cincinnati General Hospital has been found practical.

A. PRIMARY VASOMOTOR DISTURBANCES
   a. Vasoconstrictor Disturbances.
      1. Raynaud's disease.
      2. Acrocyanosis (acroasphyxia chronica; acroparesthesia; sclerodactylia).
   b. Vasodilatory Disturbances.
      1. Erythromelalgia.
      2. Acute painful osteoporosis.

Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

B. Primary Organic Disease of the Arteries

a. Traumatic (chemical and thermal).
   1. Embolism and simple thrombosis.
   2. Arterio-venous aneurysm.
   3. Phenol and all caustics.
   4. Frost bite.

b. Inflammatory (toxic).
   1. Thrombo-angiitis obliterans.
   2. Specific arteritis (syphilis, tuberculosis, periarteritis nodosa, pyogenic).
   3. Non-specific arteritis (exanthomata, typhus, typhoid, pneumonia).
   4. Non-specific arteritis (chronic toxemia, ergotism).
   5. Endarteritis obliterans (cause undetermined).

c. Degenerative changes.
   1. Arteriosclerosis (senile, diabetic and Monckeberg).

As this paper deals with the conservative treatment of the primary organic diseases of the arteries, the primary vasomotor disturbances will not be considered.

The vast majority of patients whom we see have some form of obliterative vascular disease in which there is little or no element of vasospasm.

A careful history will bring out the character and degree of pain, the time and manner of onset, personal habits and past illnesses.

The symptoms are due to lessened blood supply to the tissues of the affected extremity. In the beginning it may be only a burning sensation in the foot, with shooting pains into the muscles of the calf of the leg. There may be a sensation of numbness in various parts of the foot. In more advanced cases the patient complains of the syndrome known as intermittent claudication. Still later the pain is more severe and continues while the patient is at rest—the so-called rest pain. Subjective coldness is often a prominent symptom of organic arterial disease.

The objective findings are of greater importance than the patient's symptoms in arriving at a diagnosis. Briefly, they are absence or decrease in pulse, lowering of surface temperature, and color changes in the affected extremities, with ulceration and gangrene in the late stages.

As a rule the foregoing signs can be elicited by palpation and inspection. The oscillometer, thermocouple and x-ray are helpful.

Oscillometry determines the magnitude of pulsation in the arteries and is useful in locating the point at which the circulation is adequate in the case of organic obliteration. It does not give information as to collateral circulation.

X-ray will show calcification of the arteries, but this does not necessarily mean that the circulation is inadequate.

The thermocouple is valuable in noting minute changes in surface temperature. Observations must be made under controlled conditions of temperature and humidity.

The diagnosis of the sudden obliteration of a major artery is usually not difficult. The sudden pain, absence of pulse and cold white extremity cannot be mistaken.

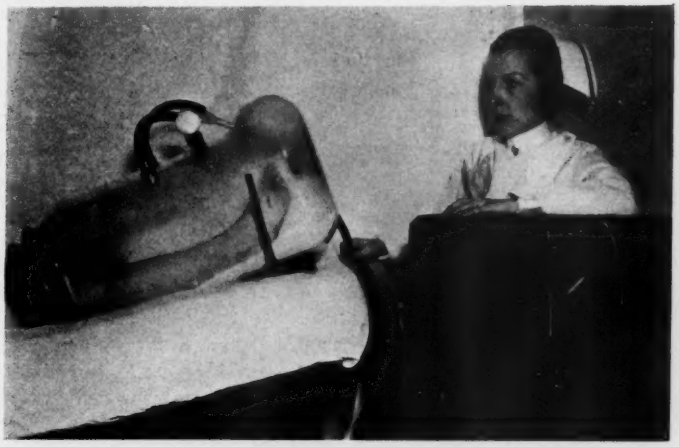


Fig. 1. The Pavaex Unit with treatment boot. Note the foot and leg is elevated to assist in the return of the venous blood.

It is difficult and often impossible to distinguish thrombo-angiitis obliterans from arteriosclerosis by means of signs and symptoms. Coldness, intermittent claudication and pain are common to both diseases. The same may be said of the color changes and arterial pulsations. All these symptoms are merely an expression of deficient circulation.

The following points are of importance: Thrombo-angiitis obliterans usually occurs in young males, with the onset of symptoms before the age of 40. Migrating phlebitis is usually present, blood pressure usually low and radial arteries soft. Albuminura is rare and there is no arcus senilis.

Arteriosclerosis is common in both men and women, usually after the age of 40. Blood pressure is often high and radial arteries are thickened. Arcus senilis is frequently present. Arteriosclerosis is also frequently associated with diabetes, and the process is similar, except that the arteriosclerosis becomes evident at an earlier age.

The principal objective sought for in the treatment of these organic deficiencies of peripheral circulation is the re-establishment of an adequate collateral circulation and the relief of pain.

While operations on the sympathetic nervous system have been helpful, when vasospasm predominates they have been disappointing in the obliterative diseases. It is true that often vasospasm plays a part in organic disease, but relieving the spasm does not affect the obliterative changes in the vessel walls. It is not logical to subject a patient to a formidable operation, such as ganglionectomy, to relieve a minor part of the cause of his circulatory failure.

The older conservative treatments of improving collateral circulation by physical methods and active exercises have been beneficial but the improvement has been of short duration.

Startling advances in the conservative treatment of obliterative vascular disease have been made since the report of Herrmann and Reid in 1933 on the results obtained by the use of PAVAEX.

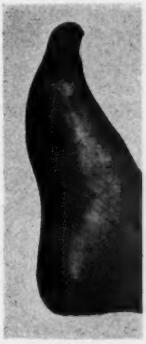
The name Pavaex is a contraction of the words passive vascular exercises. The apparatus consists of a motor driven pump and a glass chamber into which the extremity is placed. The glass boot is fitted snugly about the thigh or arm by a soft rubber cuff, and the boot is connected with the pump by means of a rubber tube. The machine runs automatically and is capable of producing any strength of negative and positive environmental pressure at any selected rate of alternation. Usually 80 mm. of mercury negative, and 20 mm. of mercury positive pressure have been found the most beneficial, and the rate of alternation is one complete cycle in about 15 seconds.

At about the same time that Herrmann and Reid reported the results of their treatment with Pavaex, Landis and Gibbon reported the physiologic effects of alternating suction and pressure upon the circulation of the normal human extremity, and later used it in pathologic conditions of the peripheral circulation. The technic employed by them differs from that used by Herrmann and Reid in that they employ sudden exchange of extreme degrees of suction and pressure. The danger of higher positive pressure is that it may contribute to the development of acute arterial thrombosis. Since positive pressure empties the capillary and venous bed of the affected extremity, so that it can be filled again during suction, it is easy to accomplish this by simply elevating the extremity during treatment.

The frequency and length of treatment varies, but on the average is from 2 to 5 hours daily. The acute arterial occlusions by operation, trauma, or embolism, should be treated intensively until ade-

quate circulation has been permanently established. Patients whose condition requires hospitalization, should have from 5 to 6 hours treatment daily, while the ambulatory patients should receive 12 to 20 hours each week.

Approximately 60 to 100 hours is necessary to develop an adequate collateral circulation in patients with obliteration of major or secondary arteries. This development of collateral circulation is brought about by causing the smaller arteries to dilate. This can be demonstrated by arteriographic studies and the palpable pulses in the genicular arteries.



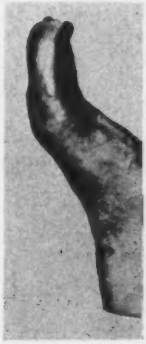


Fig. 2. Arteriosclerosis diabetic. Gangrenous ulceration on left first toe. (Case 1)
Fig. 3. Condition of foot after six weeks of passive vascular exercise treatment. Ulcer completely healed. (Case 1)

The most striking results have been obtained following the ligation of a large artery, or its occlusion by emboli or thrombosis. Pain is usually relieved in a few hours treatment, and in the majority of cases amputation has been avoided.

Frozen feet have responded promptly if intensive treatment is instituted before complete obliteration of the arterioles has taken place.

In arteriosclerosis with or without diabetes, passive vascular exercises have proven to be an efficient means of overcoming the ischemia of the extremity. Pain is relieved and trophic ulcers usually heal rapidly. Threatened gangrene may be aborted and small areas of dry gangrene may be made to demarcate with minimum loss of tissue. Massive spreading gangrene demands amputation, and good judgment must be exercised in deciding when surgery is indicated.

Recently Herrmann[9] has used local hyperthermia in conjunction with passive vascular exercises by means of a heat unit constructed in the glass boot. He reports good results in the less malignant type

of moist gangrene and cellulitis, but warns us of the necessity of carefully selecting cases.

Thrombo-angiitis obliterans is a progressive inflammatory disease of unknown origin, and so far no single etiologic factor has been found. We do believe passive vascular exercise is beneficial in the earlier stages of the disease in that it relieves pain and delays the progress of the disease by improving collateral circulation. Pavaex treatment has been disappointing in the late stages, and the good results are usually only temporary.

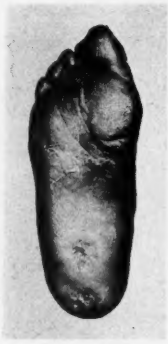


Fig. 4. Gangrenous area on left first toe and heel due to arteriosclerosis and diabetes. (Case 2)

Fig. 5. Condition of the foot after five weeks of passive vascular exercise treatment. (Case 2)

Un-united fractures and delayed union, acute painful osteoporosis and arthritis have been successfully treated by means of passive vascular exercises, and it is believed that with the combination of local hyperthermia even better results may be accomplished. The contraindications to this form of treatment are phlebitis, cellulitis or encapsulated pus.

It must be emphasized that passive vascular exercise is not a cure-all and that the older conservative methods of treatment that have proven of value should not be neglected. Briefly, the most important of these are general care of the patient and the treatment of the underlying disease responsible for his circulatory insufficiency.

All patients with vascular disease should be instructed as to the care of the feet to prevent infection. If infection does occur careful surgical attention is required. The feet should be kept warm by loose fitting socks, as hot water bottles and electric heaters are often harmful. The extremities should be horizontal and not elevated.

Postural exercises, as advised by Buerger and modified by Allen should be carried out. Tobacco in any form should be prohibited.

In the past year we have given 2850 hours of passive vascular exercise treatment to a group of 36 patients, divided as follows:—

1. ARTERIOSCLEROSIS, SENILE: 12 cases. Average age 66. All complained of some form of pain, trophic ulcers in 3, and gangrene of one or more digits in 2.

RESULTS

Marked improvement 6

Relief from pain and healing of ulcers.

Moderate improvement 3

Pain only partially relieved and ulcers healing slowly.

No improvement 3

Reasons for failure, advanced age and involvement of smaller arterioles. Amputation necessary in one case for gangrene.

2. ARTERIOSCLEROSIS, DIABETIC: 16 cases. Average age 59. Ten had trophic ulcers of the toes or some part of the feet or ankles. Beginning gangrene of the toes in 3.

RESULTS

Marked improvement 10

Relief of pain, gangrene aborted or limited, and healing of ulcers.

Moderate improvement 4

Moderate relief of pain and slow healing of ulcers.

No improvement 2

Reasons for failure: In one there was rapidly spreading gangrene, amputation necessary. In the other, treatments were refused on account of increase of pain.

3. THROMBO-ANGIITIS OBLITERANS: 4 cases. Average age 41.

RESULTS

Good 2

Both in early stages with intermittent claudication the chief symptom. Walking distance definitely increased.

Poor 2

These were far advanced. Complained of severe rest pain, and had indolent painful ulcers of feet. Pain only slightly relieved, ulcers smaller but not healed.

4. EMBOLISM—major artery: 2 cases. One femoral, one iliac.

In one case treatment began four hours after the accident, with spectacular good results. In the other case treatment was delayed 25 hours, and even then collateral circulation was established to the knee and Gritti-Stokes amputation was done.

5. FROZEN FEET: 1 case. Results good.

6. ACUTE PAINFUL OSTEOPOROSIS: 1 case. Results good.

CONCLUSIONS

Passive vascular exercise is a logical and efficient means of assisting in the establishment of an adequate collateral circulation in obliterative vascular diseases of the extremities.

The older accepted methods to promote better circulation in the extremities should be used in conjunction with this method of treatment.

REFERENCES

1. Brown, G. E.: Diseases of the Peripheral Arteries; Classification, Diagnosis, and Treatment, Pennsylvania M. J. *36:* 305-312 (Feb.) 1933.
2. Morton, J. J., and Scott, W. J. M.: The Measurement of Sympathetic Vasoconstrictor Activity in the Lower Extremities, J. Clin. Investigation *9:* 235-246 (Oct.) 1930.
3. Allen, A. W.: Recent Advances in the Treatment of Circulatory Disturbances of the Extremities. Results Obtained in Peripheral Circulatory Clinic of Massachusetts General Hospital, Ann. Surg. *92:* 931-946 (Nov.) 1930.
4. Adson, A. W.: Results of Sympathectomy in the Treatment of Peripheral Vascular Diseases, Hirschsprung's Disease, and Cord Bladder, Ann. Int. Med. *7:* 1044-1068 (Feb.) 1933.
5. Silbert, Samuel: Thrombo-Angiitis Obliterans (Buerger) XI. Treatment of 524 Cases by Repeated Intravenous Injections of Hypertonic Salt Solution; Experience of Ten Years, Surg., Gynec. & Obst. *61:* 214-222 (Aug.) 1935.
6. Herrmann, L. G., and Reid, M. R.: The Pavaex (Passive Vascular Exercise) Treatment of Obliterative Arterial Disease by Means of Intermittent Negative Pressure, J. Med. *14:* 200-204 (June) 1933.
7. Flothow, P. G., and Swift, G. W.: Surgery of the Sympathetic Nervous System: Review of 100 Sympathetic Ganglionectomies, Am. J. Surg. *21:* 345 (Sept.) 1933.
8. Landis, E. M., and Gibbon, J. H., Jr.: The Effects of Alternate Suction and Pressure on the Blood Flow to the Lower Extremities, J. Clin. Investigation *12:* 925-961 (Sept.) 1933.
9. Herrmann, L. G.: Nonoperative Treatment of Inadequate Peripheral Distribution of Blood; Passive Vascular Exercises and Local Hyperthermia. J. A. M. A. *105:* 1256 (Oct. 19) 1935.

TUMORS OF THE TRACHEA

CHEVALIER JACKSON, M. D.
Philadelphia

TUMORS of the trachea have been given full and deserved attention by pathologists[1, 2]; their histology has been extensively studied and classifications have been based thereon. From force of habit discussions on tumors usually start with a classification into benign and malignant growths. There is no objection to this provided it be recognized that all tumors cannot always be placed in either of these two classes. A third class must be made, namely, borderline growths. Furthermore we must not fall into the error, often seen in the literature, of using the word innocent as synonymous with benign. A growth in the trachea may be histologically benign and yet it may kill the patient by asphyxia. It is the purpose of the present paper to consider the subject from a purely mechanical viewpoint.

Classification. From the mechanical point of view the most important classification is endotracheal, murotracheal and peritracheal. Another point in mechanical classification is into obstructive and nonobstructive growths. A third feature of classification, and it is a very important one, concerns the region affected, namely, (a) the cervical trachea and (b) the intrathoracic trachea. Between these two there is a very important region namely, the upper thoracic aperture. This very important region presents the mechanical feature of a rigid bony ring through which pass not only the breath of life but, additionally, vital channels of communication between the brain and the thorax. Because of the rigidity of the ring filled as it is by tissues, there is no room for a tumor without compression between the mass and the ring.

Growths involving the *party-wall between the esophagus and trachea* should be regarded as a distinct class because of a number of clinical features. In the first place they are nearly always accompanied with recurrent paralysis, owing to the close relationship of this party-wall to the vagus and to the recurrent fibers therein. We have seen many cases in which there was a recurrent paralysis but no other symptoms of the pressure on the vagi in cases in which there was clearly no involvement of the recurrent nerve itself. We believe that it is an established clinical fact according to our observations, that any pressure on the vagus above the giving off of the recurrent laryngeal nerve may produce a laryngeal paralysis long

Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

before there is any evidence of injury to any other set of nerve fibers in the vagus. The next most important matter in connection with party-wall growths is the maintenance of a proper esophageal lumen after the removal of the tumor. The utmost care is required, likewise in the case of benign growth, to separate the esophagus, even if nothing but the mucosa is left, without opening into the esophagus itself. It is always desirable not to be required to suture the esophageal wall. Usually this will not be necessary if in the dissection the esophageal wall is not encroached upon sufficiently to destroy this mucosa even in a very limited area. If however, it is impossible to get the growth away without removing esophageal mucosa, then the utmost care should be taken to leave the greatest possible amount of the periphery of the esophageal wall. If sufficient mucosa be left to form a lumen, however small that lumen may be, esophagoscopic methods will permit of dilatations to a point where the patient can have perfectly normal swallowing. Even a continuous vertical strip of mucosa will help. If the mucosa is destroyed throughout the entire periphery of the esophagus, even though it be a very narrow band, total atresia is sure to follow unless the patient is placed promptly in the hands of the esophagoscopist for the maintenance of a lumen. The dilatation of any sort of fistula is easy and safe; the perforation of an esophageal atresia is a very dangerous procedure.

Nonobstructive growths as a rule are simply growths that have not enlarged sufficiently to cause obstruction under ordinary conditions. The lumen of the trachea is more than ample to supply the need of air, in fact it can transmit more air than can enter through the glottis under atmospheric pressure. Its area of cross section is more than twice that of the widely open glottis. For this reason very small growths may not give signs of tracheal obstruction.

Obstructive Tumors. Endotracheal tumors obstruct by their bulk in the lumen of the trachea. Murotracheal and peritracheal growths obstruct by crowding the wall inward until the lumen of the trachea is encroached upon sufficiently to cause obstruction.

All three types of growth may cause any one of the four types of valvular obstruction. These have been previously described[7, 8]. It will be necessary here only to name them: 1, Stop-valve obstruction, in which air cannot pass in either direction; 2, by-pass valve obstruction, in which the air can pass both in and out but in diminished quantity; 3, check-valve obstruction permitting the ingress but not the egress of air, producing emphysema of both lungs; 4, check-valve obstruction permitting air to escape from the lung but

preventing ingress, producing atelectasis; if the obstruction is complete as to admission of air, asphyxia promptly supervenes.

All of the foregoing types of obstruction are easily understood by anyone familiar with hydraulics and aerodynamics. There is one type of obstruction however, that we have discovered with the bronchoscope [7, 8] that seems never previously to have been recognized, namely, the *expansile check valve*. It has no parallel in hydraulics or aerodynamics. It is due to the rhythmic increase and decrease in size of lumen in inspiration and expiration respectively, under the influence of the thoracic bellows. So long as the lumen remains unobstructed there is of course, no valvular action in the trachea. When however, the lumen is encroached upon to the extent that it is obliterated before the end of the expiratory phase, air is trapped below the point of narrowing. On inspiration air is taken in; but all of it cannot escape on expiration; the progressively accumulated air below causes an obstructive emphysema. This constitutes the expansile check valve. The *flapper type* of check valve commonly occurs with the pedunculated tracheal tumors. The *ball valve type* of check does not occur in the trachea in cases of tumor unless the growth should become detached or a ball of secretions should be formed. A *compressive peritracheal growth* may produce a check-valve obstruction just as efficiently as endobronchial growth; we have seen many such cases.

There is another mechanism of obstruction that must not be overlooked, namely, *recurrent paralysis*. If only one recurrent nerve is affected the obstruction will produce no dyspnea except on exertion. It may also produce no effect whatever on the voice. In most instances however, there is more or less phonatory impairment for a time. Just as soon as proper adjustments with the normal cord are made, hoarseness usually disappears. In fact we have known a number of public speakers, ministers, lawyers, judges and politicians who have done very effective public speaking notwithstanding that one cord was paralyzed. Paralysis of one or both cords may be present in a case of a tracheal tumor that is not producing any other sign or symptom to lead to suspicion of its presence. Over and over again patients have come in to the clinic complaining only of *hoarseness*, yet bronchoscopic examination has demonstrated the presence of a tumor in the trachea.

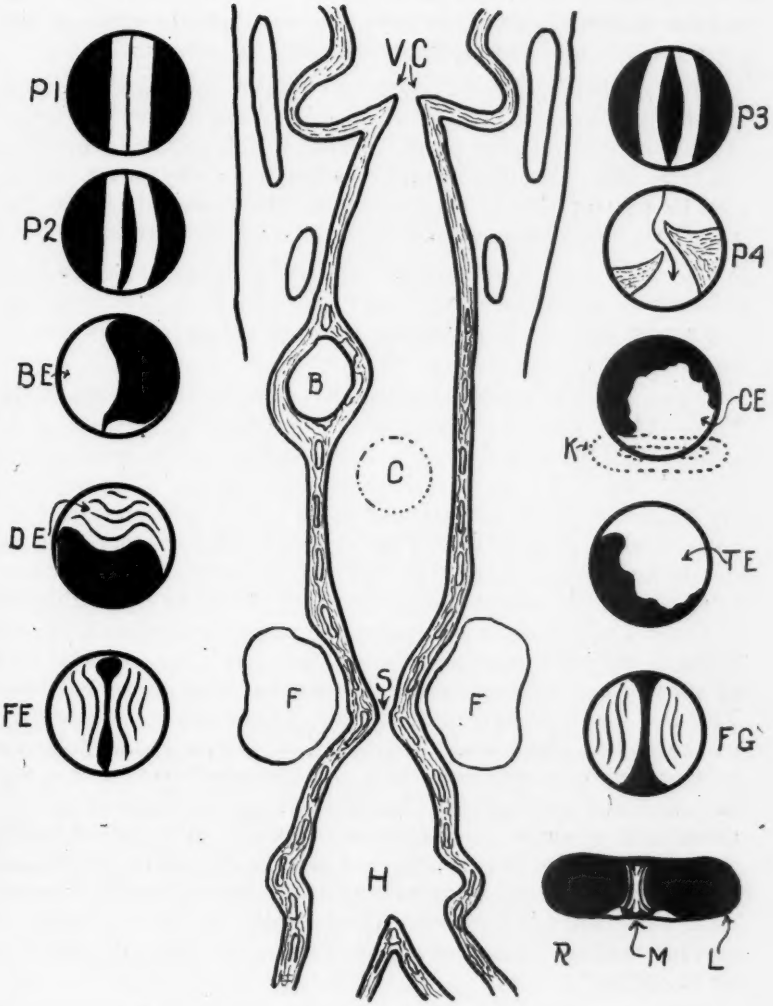
Varities of Tracheal Tumors. The records of the bronchoscopic clinic show the following list of tumors: angioma, hematoma, adenoma, myoma, myxoma, fibroma, fibrolipoma, fibromyxoma, myxomatoid edematous polyps, lymphoma, ecchondrosis, ecchondroma, osteophytoma, osteoma, chondrosteoma, retention cysts,

epidermoid cyst, aberrant thyroid tumor, goiter, hypertrophic thymus gland. Of the infective diseases tumor-like endobronchial formations are found bronchoscopically in tuberculosis, actinomycosis, blastomycosis, syphilis and the common suppuration due to pyogenic organisms[13, 14, 17]. These tumor-like granulomas are obstructive and tend to perpetuate suppuration. Gumma has been found bronchoscopically a number of times[13]. The most common peritracheal growth is, of course, goiter. The most common endotracheal tumor is papilloma, and next in frequency comes carcinoma.

Of the unquestionably malignant growths in the trachea and in the surrounding region the most frequent in our experience has been carcinoma, next in frequency comes sarcoma and the least frequent endothelioma and quite a variety of borderline tumors that in some instances are malignant and in others not.

Symptomatology. The most important of all symptoms of tracheal growth is a wheeze heard at the open mouth. This we have called the asthmatoid wheeze because of the similarity of sound[17]. The mechanism of this sound is simply a mechanical obstruction to the passage of air in a fricative way. There is no occasion to wander off into the colossal literature on the etiology and mechanism of asthma to account for this very simple phenomenon of a column of air being set in vibration by passing through a narrowed lumen. The asthmatoid wheeze is usually present for a long time before the second most important symptom develops.

Schematic illustration of the pathologic mechanism of tracheal tumors, and tumor-like conditions, in our series of cases. Dyspnea, stridor and wheezing which are all prominent features of tracheal tumors were in some cases due to paralysis of the vocal cords (VC), the lumen of the trachea not being encroached upon by the peritracheal growth. The other cases here diagrammed are as follows: B. A tumor (chondroma) in the wall of the trachea encroached on the tracheal lumen as indicated in the schema of the endoscopic appearance (BE). From the location, C, there sprang from the posterior wall a leiomyoma (CE) that originated in the esophagus (K). From near the same level, in another patient, there sprang from the anterior wall an aberrant endotracheal thyroid-gland tumor (TE). In another patient an aneurysm mounded in at about this location (DE). A peritracheal adenoma (F) compressed the trachea so as to form a syrinx (S); on forced expiration a loud phonation came out of the bronchoscope and the syringeal margins were seen to vibrate as they set the column of air in vibration (FE). The following inspiration would open this syringeal glottis (FG). H. An anteroposteriorly compressive tumor (lymphoma) just above the bifurcation produced a tracheal lumen of elliptic outline beneath which could be seen the carina (M) and the right (R) and left (L) bronchial orifices. In our experience, when recurrent paralysis occurs during operation on a patient with tracheal tumor, the vocal cords may be in four different positions as follows: The cords may be in the median position (P1); the patient will asphyxiate unless promptly relieved. If one cord is cadaveric (P2) enough air will enter barely to sustain life. If both cords are in the cadaveric position (P3) the patient gets enough air to prevent cyanosis and even indrawing at the suprasternal notch; this primarily cadaveric position may be succeeded by the median position (P1) threatening asphyxia. In some instances the cords may appear in the mirror as in P1, but a difference in level will permit air to enter as indicated by the dart in P4.


P1
P2
BE
DE
FE
VC
B
C
F
S
F
H
P3
P4
CE
K
TE
FG
R
M
L

The latter is dyspnea; often nocturnal paroxysms of dyspnea, due to accumulation of secretions around and below the tumor. When the increase in size of the growth encroaches sufficiently on the lumen a wheeze is produced; at this stage the dyspnea becomes constant and the nocturnal paroxysms more severe. If the progress of the growth is not arrested asphyxia is usually the termination.

Of the other symptoms cough and expectoration are the most important. These may be relatively insignificant or they may occur in the form of strangling paroxysm ending with getting up more or less secretions. Hemoptysis may occasionally be a symptom. Fever may be present due to suppuration of the growth itself or the pulmonary suppuration secondary to the tracheal obstruction.

One of the most curious and interesting things in our records referable to symptomatology is the relatively large number of patients who date their symptoms from an attack of influenza or the grippe.

Diagnosis. The symptomatology is not sufficient for a diagnosis. There is only one way positively to determine the presence of an endotracheal growth, or of a compression due to peritracheal growth, and that is by bronchoscopic examination. The roentgen ray should be used in all cases and often reveals information of the utmost value. If shadows of the tracheal walls do not stand out clearly with a negative shadow showing the lumen, the outline may be strengthened and rendered very strong indeed by the instillation of lipiodol. That there is a tumor present in the trachea is readily determined by the bronchoscopic examination. The particular kind of growth can be conclusively decided by *bronchoscopic biopsy.* This is a simple procedure and yields definite and invaluable diagnostic results. The bronchoscopist will of course, use his judgment in the taking of a specimen. In a case of a small growth it is best to remove the entire growth. In cases of larger growths or multiple growths, a specimen alone may be removed. In a case of benign growth of small extent it is best to remove the entire growth and subject it to histologic examination. If the bronchoscopic diagnosis as to benignancy is confirmed histologically, the growth being removed, nothing further is required in most cases. Papilloma is an exception.

The differential diagnosis between tracheal tumors and *asthma* can be made only by bronchoscopic examination. Our records show that every case of bronchial tumor that did not cause external bulging has been mistaken for asthma because of the wheezing sound. Of course, by this we do not mean to imply that the diagnosis of asthma rests solely on the wheeze; and certainly it would be

very unjust to assume that the wheeze is a large or important element in the diagnosis of asthma in an asthma clinic or in the work of the internist; but it seems that the busy practitioner whose time is taken up largely with very serious acute illness may make the diagnosis of asthma when there is little to support such a diagnosis other than the wheeze. Contributing to this diagnostic confusion are the nocturnal paroxyms of dyspnea due to accumulation of secretions as above mentioned. Another source of confusion is the fact that the patient may be allergic, reacting to sensitization tests and contacts. It is a clinical fact that our records show that some physician told the patient he had asthma in almost every case that did not at the time of his examination show a tumor externally. Another erroneous diagnosis often made is that of thymic compression. There are many forms of laryngeal stenosis that are attributed to thymic compression when the thymus gland is not to blame. In the presence of a wheeze and dyspnea a differential diagnosis must be made as among (a) recurrent paralysis; (b) endotracheal tumor; (c) compressive peritracheal tumor.

In case of tracheal growth above the level of the suprasternal notch the presence of the tumor can be determined by palpation and the skilful fingers of the surgeon will very often determine the kind of tumor. It cannot be too strongly emphasized however, that this skilful diagnosis does not lessen the necessity for a *mirror examination in every case*. Over and over again we have seen the surgeon blamed for a recurrent paralysis that had existed in one cord for probably many years prior to his operation. In some instances the second cord was paralyzed at or soon after operation. In other instances the patient has come in complaining that a surgeon paralyzed one cord but that the condition was never discovered until it was found in the course of a mirror examination a shorter or longer time after operation. Additionally, for the welfare of the patient, if one cord is paralyzed the fact should be known beforehand, so that proper precautions can be taken for dealing with impending asphyxia that would occur if the remaining cord should be paralyzed at operation. A postoperative paralysis may be cadaveric at the start and may become median afterward; a reversal of Semon's rule. One side may be cadaveric, the other median. Anomalies of innervation are probably responsible.

Syphilis is of course to be routinely excluded. We have seen a number of cases in which gumma of the trachea simulated a tumor. The serologic reaction was strongly positive, and heavy doses of potassium iodid and mercury caused the recession of the syphilitic tumor. In the more recent cases bismuth sodium tartrate by injection has been used.

In all cases of tracheal tumor diagnostic esophagoscopy should be done, whether there are any symptoms referable to the esophagus or not, because of the vital surgical importance of esophageal involvement.

Treatment. Benign endotracheal tumors of limited extent require only bronchoscopic removal. This will be curative with all tumors in this class with the sole exception of multiple papillomas. These may not recur at the site from which they were removed but new growths may appear at any site, not only in the trachea but they may appear elsewhere, that is to say in the bronchi, in the larynx, pharynx, fauces, nose or even the mucosal junction with the skin at the interior nares or the lips. These are of course new growths not repullulation of the primary process. Their appearance in these remote locations however, indicates plainly the futility of attempting radical removal of the base at the original site in the trachea. It is improbable that the removal of the endotracheal growth has anything to do with the appearance elsewhere; there is nothing to justify an opinion that dissemination is caused thereby. Experience with hundreds of cases of multiple papillomas in the trachea has convinced us that the best plan of procedure is superficial removal repeated often enough to keep the lumen open. In some instances we have seen the roentgen ray arrest the growth in one location; but obviously it cannot be applied indiscriminately to all of the locations that may be invaded. In any event it requires a dosage almost equal to that required for malignant disease. Such dosage always involves the risk of perichondritis and chondral necrosis. This is of course fully understood by radiologists who may be depended upon to guard against a disaster due to the exquisite sensitiveness of tracheal as well as laryngeal cartilages to radiation. We had many experiences in dealing with intractable cicatricial stenosis of the trachea resulting from radiation in the early days of the roentgen ray before this sensitiveness of the cartilages was realized.

It is, of course, unnecessary, and indeed it would be quite presumptuous for me before this Congress to discuss the surgery of goiter. With all due humility however, it should be said that every goiter operation should be preceded by a mirror examination of the larynx by an expert in the determination of defects of laryngeal motility. These are often very slight and very easily overlooked except by those well trained in this kind of work. Forty years ago I urged very strongly at a congress of surgeons that in every operation on the neck, and especially if done for goiter, preoperative mirror examination of the larynx. This suggestion at the time was ridiculed as entirely superfluous. Since that time I have seen hundreds

of cases in which the surgeon thought there was nothing wrong with the motility of the larynx because the patient had a good voice, yet a unilateral paralysis was present.

Peritracheal growths other than goiter should be dissected free from the trachea. A few such growths have capsules that render this dissection easy. Most of the growths however, are not strictly speaking encapsulated. Peritracheal malignant growths metastasize to the mediastinal glands so early that in most cases roentgen-ray treatment will be found better for the patient than any form of operation. It is true that the choice will depend upon the degree of malignancy; but in any event the roentgen ray is indicated to seal the lymph channels, as early as the diagnosis can be made.

The treatment of *growths involving the esophagus* are in a class by themselves. In no other structure in the neck is the surgeon confronted with as great difficulty in obtaining a satisfactory result as in these cases of esophageal involvement. A growth even though benign starting in the esophageal walls and reaching the point where the trachea becomes involved presents a problem for the surgeon that is serious out of all proportion to the histologic benign diagnosis. Surgically the esophagus is one of the most intolerant organs in the body. The chief advance that has been made in the last forty years has been the development of the art of esophagoscopy to the point where any benign postoperative stenosis can be dilated so as to give the patient a perfect swallowing function. This can be done safely if it be done slowly. Rapid dilatation of any form of esophageal stenosis will be attended with a high degree of mortality. By way of contrast very close to 100 per cent of permanent cures can be obtained in benign stenosis by esophagoscopy, with no mortality whatever if the work be done patiently and slowly; and provided, especially, that the case is not one of total atresia. The latter condition introduces a large percentage of mortality; there is however, no need of permitting a total atresia to occur. Some sort of a lumen could be maintained if esophagoscopic means are instituted early. In all operations for tracheal growth involving the esophagus the utmost care should be taken to preserve intact as great a part of esophageal wall as the circumstances may permit. Any vestige of esophageal epithelium is invaluable in reconstruction of a swallowing tube. Metaplasia may convert the ciliated columnar epithelium into squamous; but this seems to function very well.

Complications

Bilateral Recurrent Paralysis. Paralysis of one cord is not a great disaster because the airway is ample and the voice if impaired

will very quickly improve to some extent; in time it will usually improve very greatly. In bilateral recurrent paralysis the voice is usually good; but the dyspnea may result in asphyxia. This has happened on the table in the experience of most surgeons. Many if not most deaths that had been attributed to other causes, such as central lesions, hypertrophic thymus, cardiac complications and so on, were really cases of bilateral laryngeal paralysis. When bilateral recurrent paralysis occurs on the operating table one of three things may happen. Namely: 1, the patient may promptly asphyxiate; 2, the patient may get along on a diminished amount of air; 3, quiet and satisfactory respiration may be restored by an alert anesthetist inserting an intratracheal insufflation anesthesia tube.

To understand conditions 1 and 2 it is necessary to remember that bilateral recurrent paralysis occurs in three forms; (a) the cords assume the midline position with edges in contact or nearly so; in this position the greater degree of inspiratory effort the less air enters because the angle at which the cords are set results in the pressure closing the cords tightly when they are paralyzed; in other words the cords are paralyzed shut and held tightly in apposition by the air pressure. (b) The cords may be on a different level permitting air entrance between them just as in the baffle plate used in mechanics. This difference in level is probably due to the irregular pull of some of the unaffected muscular fibers or to irregularities of position of the arytenoids, or irregularities of the articular surfaces of the cricoarytenoid joint. (c) The cords may at once assume the cadaveric position. This position ordinarily is not assumed until paralysis has been present for a long time; but in certain cases it occurs immediately. It is a curious thing that when both recurrent nerves are completely severed the paralysis is usually not cadaveric as to the position of the cords. The midline position is much more common. We have seen, however, in a number of cases, a cadaveric position of the cords followed by a median position of the cords. This is an exception to the rule of Semon that the postici or abductor muscles are first affected, the tensors secondarily and the adductors thirdly.

The treatment of the complication, bilateral recurrent paralysis, is clearly indicated. When the paralysis occurs on the table the quickest and best thing to do is for the anesthetist to put in an anesthetizing tube. Of course, a bronchoscope may be used if one should be at hand; but every anesthetist has a direct laryngoscope and an anesthetizing catheter that he can promptly insert into the trachea in any case of respiratory difficulty. If the anesthetist is not so equipped the next best thing is for the operator to do a tracheotomy. As the trachea is usually exposed during the operation that

caused bilateral recurrent paralysis it is quite simple for the surgeon to make an incision in the trachea and insert a tracheal cannula with which every operating room is or ought to be equipped. Having tided over the emergency a tracheotomy will be required in the cases in which this has not been done as part of the treatment of the emergency on the table. Under all circumstances the *tracheotomy should always be low.* A high tracheotomy almost invariably introduces a condition of subglottic stenosis. In order to emphasize this point it may here be stated in passing that in most cases the cicatricial stenosis is due to high tracheotomy and not to the primary acute stenosis for which the tracheotomy was done[18].

Next comes the matter of decannulation. This has been fully covered in previous publications[4, 5, 6, 19].

Looking at the matter broadly, regardless of the conditions present, bilateral recurrent paralysis that persists longer than a few weeks will be permanent. Degeneration of the nerve tends to follow very promptly after the onset of a recurrent laryngeal paralysis. Regardless of whether the condition is (a) due to pressure of the growth prior to operation, or (b) due to injury to the nerve during the operation, (c) cicatricial contraction afterward, the result is always the same; that is to say that if movement does not reappear within a few weeks it will never come back. In the diagnosis of recurrent paralysis it is necessary always to exclude impairment of motility due to arthritis or fixation of the cricoarytenoid joint. This is done by direct laryngoscopy; passive mobility in lateral pressure with a probe is always present in paralysis.

Palliative Treatment of Malignant Disease of the Trachea. When a patient with carcinoma, sarcoma, endothelioma or malignant lymphoma of the trachea comes in too late for any hope of surgical cure recourse must be had to palliative measures. First and foremost it must not be forgotten that after a palliative tracheotomy is done our duties are not finished; tracheotomy is only a means toward the end; which is the piping of air down into the lung. By means of the long tracheal cannulas this can be done just as long as there remain functioning enough air cells to carry on their vital duties. It is sometimes asked "What is the use?". In answer to this it must be said that while palliative operation cannot cure the malignant disease it can prevent the patient dying of asphyxia. It is repugnant to all surgical instinct to stand by and allow a patient to asphyxiate when the surgeon knows that he might give the patient many months of life by the operation of tracheotomy followed up by careful after-treatment to insure that the air is really getting to the lung. Moreover, in many of these

cases if the airway be maintained the roentgenologist can have a free hand in the irradiation. Many times we have been surprised at the complete arrest of a malignant disease in or close to the trachea by efficient methods of radiation. In a large percentage of the cases the growth has returned; but the same thing may be said of any operation for malignant disease. Notwithstanding this well established clinical fact, surgery should always be resorted to in all operable cases.

Sequelae. Cicatricial stenosis from the removal of a peribroncheal growth occasionally occurs. It can be cured by peroral endoscopic dilatation in most cases; a few cases may require laryngostomy. Stenoses of the esophagus are referred to above in connection with growths of the party-wall. If removal of a considerable section of the length of the esophagus is necessary gastrostomy will be required.

Conclusions

1. The symptoms of tumors of the trachea are: (a) wheezing respiration; (b) nocturnal attacks of dyspnea; (c) dyspnea on exertion; (d) asphyxia, unless averted.

2. In case of tracheal tumor the dyspnea may be due to: (a) recurrent paralysis; (b) endotracheal tumor; (c) compressive peritracheal growth. One, two or all three of these conditions may be present in any given case; differentiation is by direct laryngoscopy and bronchoscopy.

3. Diagnosis is by (a) palpation; (b) roentgen ray; (c) biopsy; (d) bronchoscopy; (e) bronchoscopic biopsy; (f) in some instances, esophagoscopy.

4. Curable cases require bronchoscopic removal of endotracheal growths; external surgery for peritracheal growths.

5. In cases of esophageal involvement utmost care should be taken to leave the esophageal mucosa intact if possible; even a vertical strip of mucosa uniting the upper and lower segments will help the esophagoscopist to create an adequate and permanent esophageal lumen. The same principle applies to the tracheal lumen, but here we have laryngostomy to fall back upon.

6. Lymphoid tumors are best treated by irradiation.

7. In cases of inoperable malignant growths the most important duty of the surgeon is to pipe the air down into the lung, using a well-fitted cannula of sufficient length for the purpose; if long the inner portion of the cannula should be flexible.

8. Finally, the diagnosis of asthma in any case should be only tentative until after bronchoscopy has demonstrated the absence of tracheal tumor. This rule applies whether the patient is allergic or not. All is not asthma that wheezes.

REFERENCES

1. D'Aunoy, R., and Zoller, A.: Primary Tumors of the Trachea, Arch. Path. *11:* 589 (April) 1931.
2. Ewing, James: Neoplastic Diseases, a Treatise on Tumors, Philadelphia: W. B. Saunders Co., 1922.
3. Funk, E. H.: Medical Aspects of New Growths of the Lung, Proc. Assn. Am. Phys., 1930.
4. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, St. Louis. The Laryngoscope Co., 1915. (This book is out of print, but is in most libraries. The French translation entitled Endoscopie et Chirurgie du Larynx is still obtainable from the publisher, Gaston Doin, Paris.)
5. Jackson, Chevalier, and Jackson, Chevalier L.: Surgery of the Larynx, Trachea and Endoscopic Surgery of the Bronchi, in Dean Lewis' Practice of Surgery, Chap. 7, Vol. 4, pp. 1-232.
6. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoscopy, Esophagoscopy and Gastroscopy, ed. 3, Philadelphia: W. B. Saunders Co., 1935.
7. Jackson, Chevalier: The Mechanism of Physical Signs, with Especial Reference to Foreign Bodies in the Bronchi, Am. J. M. Sc. *165:* 313-320 (March) 1923.
8. Jackson, Chevalier: The Mechanism of Physical Signs in Neoplastic and Other Diseases of the Lung, with Especial Reference to Atelectasis and Emphysema, J. A. M. A. *95:* 639-644 (Aug. 30) 1930.
9. Jackson, Chevalier, and Jackson, Chevalier L.: Benign Tumors of the Trachea and Bronchi with Especial Reference to Tumor-like Formations of Inflammatory Origin, J. A. M. A. *99:* 1747-1753 (Nov. 19) 1932.
10. Jackson, Chevalier: Malignant Growths of the Lung, Bronchoscopic Diagnosis, Arch. Otolaryng. *12:* 747-752 (Dec.) 1930.
11. Jackson, Chevalier: Malignant Disease of the Lung, Opening of a Discussion at the Medical Society of London, Nov. 10, 1930, publ. in the Proc. of that year. Abstr. in Lancet *2:* 1071 (Nov. 15) 1930.
12. Jackson, Chevalier: La Diagnosi Broncoscopica della Affezioni Neoplastiche, Ras. Inter. di Clinica E Terapia, Anno 8, n.10, 1927.
13. Jackson, Chevalier: Suppurative Diseases of the Lung; Report on a Series of Bronchoscopic Observations, Proc. Roy. Soc. Med. (Occas. Lect.) *24:* 1-24 (Nov.) 1930.
14. Jackson, Chevalier: Endoscopic Treatment of Suppurative Diseases of the Bronchi and Lungs, Postulates for Discussion. Résumé supplementary in Spanish, French, Italian, German to a rapport on the same subject at the Sec. Inter. Oto-Rhino-Laryngol. Congress, Madrid. (Sept.) 1932.
15. Jackson, Chevalier L., and Konzelmann, F. W.: Bronchial Carcinoma, J. Thor.-acic Surg. *4:* 156 (Dec.) 1934.

16. Jackson, Chevalier; Jackson, Chevalier L., and Vialle, Jacques: La bronchoscopie en pathologie bronchopulmonaire, Monographies Internationales, Delmas, Bordeaux, France, 1936.

17. Jackson, Chevalier: A New Diagnostic Sign of Foreign Body in Trachea or Bronchi the "Asthmatoid Wheeze", Am. J. M. Sc. *156:* 625 (Nov.) 1918.

18. Jackson, Chevalier: High Tracheotomy and Other Errors the Chief Causes of Chronic Laryngeal Stenosis, Surg., Gynec. and Obst. *32:* 392 (May) 1921.

19. Jackson, Chevalier: Ventriculocordectomy: New Operation for Cure of Goitrous Paralytic Laryngeal Stenosis, Arch. Surg. *4:* 257-274 (March) 1922.

20. Lord, Frederick: Diseases of the Lungs and Pleura, ed. 2. Philadelphia: Lea & Febiger, 1925, p. 585.

21. Tilley, Herbert: From Observations on the Clinical Significance of Paralysis of the Vocal Cord, J. Laryng. Rhinol. & Otol. (June) 1916.

ACUTE PERITONITIS

Some Causes and Treatment

A. STREET, M. D.
Vicksburg

This discussion is based on a review of the records of patients treated for acute peritonitis at the Vicksburg Sanitarium during the ten year period from 1926 to 1936. The management of peritonitis developing secondary to disease of the uterus and adnexa belongs to the realm of gynecology: I have omitted therefore patients having peritonitis following abortion, puerperal infection, gonorrheal salpingitis, complicated new growths or other diseases of the female genital organs. The study of these records was directed principally toward determining the source of the peritonitis, the cause, the treatment and the result.

Classification. Two patients with aseptic peritonitis due to intraperitoneal rupture of the urinary bladder were seen and operated upon early before infection had developed.

All the other cases were of the infectious type. According to the extent of the process, they were classed in the following four groups:

1. *Local Peritonitis.* Peritoneal inflammation confined to the immediate vicinity of the initial focus and not apparently spreading.

2. *Spreading Peritonitis.* Free peritoneal inflammation, moderately spread from the site of the initial focus and with no successful walling off of the process.

3. *Diffuse Peritonitis.* All cases of diffuse widespread free peritoneal inflammation. This group is the one of so-called general peritonitis, a term which I have avoided because, although the patients had the symptoms and operative evidence of general peritonitis, I did not see all of the peritoneum and could not be certain how general the inflammation was.

4. *Localized Abscess.* Cases of walled-off suppuration in the peritoneal cavity.

Patients were classified in these groups according to the diagnosis of their condition at the beginning of treatment. This was determined usually by the symptoms and the operative findings. If at operation a patient was observed to have local peritonitis, and then

Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

following the operation developed diffuse peritonitis and died, he would be classed as a failure in treatment of local peritonitis. Peritonitis purely secondary to operative procedures is likewise excluded.

Causes observed. There were 597 cases of peritonitis. They were caused by the following conditions:

TABLE 1

CAUSES OF ACUTE PERITONITIS

Cause	*Cases*	*Recovered*	*Died*	*Per Cent*
Appendicitis, acute*	443	424	19	4.28
Obstruction, acute intestinal	61	47	14	22.9
Diseases of gallbladder and liver ducts	47	42	5	10.63
Wound of abdomen, gunshot	12	8	4	33.3
Peptic ulcer	8	6	2	25.0
Wound of intestine, lacerated	7	2	5	71.4
Diverticulum of large intestine	5	4	1	20.0
Fracture of pelvis	4	4	0	0
Diverticulitis, Meckel's	3	3	0	0
Abscess of liver	1	1	0	0
Carcinoma of sigmoid colon	1	1	0	0
Typhoid fever	1	0	1	100.
Thrombosis, mesenteric, primary	1	0	1	100.
Pancreatitis, acute	1	1	0	0
Ulcer of colon, amebic	2	2	0	0
Total	597	545	52	8.74

* During the period of time reviewed, there were 923 cases of acute appendicitis, with or without peritonitis. Of these cases, 19 died, a mortality, in acute appendicitis, of 2.05 per cent.

According to this classification the cases were divided as follows:

TABLE 2

TYPES OF ACUTE PERITONITIS

Cause	*Cases*	*Recovered*	*Died*	*Per Cent*
Acute local peritonitis	408	401	7	1.7
Spreading peritonitis	37	36	1	2.7
Diffuse peritonitis	107	67	40	37.28
Local abscess	45	41	4	8.88

In reviewing the etiology I find no cases identified as so-called primary peritonitis of the pneumococcus or streptococcus type, although according to published reports[1, 2] such cases are not rare following respiratory infections, scarlet fever and measles. Bren-

neman[3] has observed a case of fatal peritonitis following rupture of a suppurating mesenteric lymph node. We have seen numerous cases of acute mesenteric lymphadenitis[4] occurring in children and young adults, but so far have seen no peritonitis resulting.

Treatment. The proper treatment of acute local and early spreading peritonitis is with few exceptions prompt operation for relief of the cause. This is generally accepted and requires no comment. If we could time our operative treatment as we desire, we would do all of these operations in the early stage of the disease. We get many patients early and operate with excellent results. However, in spite of the earnest attempts of the profession to educate the public, the patient too often manages the treatment himself during the early stages, and accelerates disaster by taking among other remedies repeated doses of cathartics.

The treatment of acute diffuse peritonitis is a surgical problem; with few exceptions it will involve operation. However, operation is often not the patient's first and most urgent requirement. Treatment may be divided into preoperative, operative and postoperative phases.

Preoperative Treatment. Not infrequently we see brought to the hospital after three or more days of illness a patient who presents a picture of markedly disordered physiology. He is dehydrated from vomiting and inability to assimilate his attempted fluid intake. The absorbing portion of his intestinal tract is paralyzed. Fluid which should pass into the lower portion of the small intestine and colon for absorption is being regurgitated into the upper gastrointestinal tract, where the upper mucosal, biliary and pancreatic secretions are added to it and the mixture is spilling out of the overdistended stomach. The loss of this fluid involves also the loss of other essential elements, particularly chlorides[5]. The patient has been in pain and has been having fever. His reserve energy is depleted. The intestinal wall is infiltrated with toxic inflammatory products as the infection extends to the subperitoneal tissues, and the pressure within the lumen of the gut impairs the intestinal circulation of blood. This, together with a disordered intestinal nerve supply, results in loss of tone. As the abdominal distention increases the patient is further handicapped by respiratory difficulty due to pressure on the diaphragm.

A patient in this condition is obviously not a favorable one to withstand the additional strain of an abdominal operation. However, if we bear in mind the principal factors responsible for his embarrassment, and then remember and use the physiologic meas-

following the operation developed diffuse peritonitis and died, he would be classed as a failure in treatment of local peritonitis. Peritonitis purely secondary to operative procedures is likewise excluded.

Causes observed. There were 597 cases of peritonitis. They were caused by the following conditions:

TABLE 1

CAUSES OF ACUTE PERITONITIS

Cause	*Cases*	*Recovered*	*Died*	*Per Cent*
Appendicitis, acute*	443	424	19	4.28
Obstruction, acute intestinal	61	47	14	22.9
Diseases of gallbladder and liver ducts	47	42	5	10.63
Wound of abdomen, gunshot	12	8	4	33.3
Peptic ulcer	8	6	2	25.0
Wound of intestine, lacerated	7	2	5	71.4
Diverticulum of large intestine	5	4	1	20.0
Fracture of pelvis	4	4	0	0
Diverticulitis, Meckel's	3	3	0	0
Abscess of liver	1	1	0	0
Carcinoma of sigmoid colon	1	1	0	0
Typhoid fever	1	0	1	100.
Thrombosis, mesenteric, primary	1	0	1	100.
Pancreatitis, acute	1	1	0	0
Ulcer of colon, amebic	2	2	0	0
Total	597	545	52	8.74

* During the period of time reviewed, there were 923 cases of acute appendicitis, with or without peritonitis. Of these cases, 19 died, a mortality, in acute appendicitis, of 2.05 per cent.

According to this classification the cases were divided as follows:

TABLE 2

TYPES OF ACUTE PERITONITIS

Cause	*Cases*	*Recovered*	*Died*	*Per Cent*
Acute local peritonitis	408	401	7	1.7
Spreading peritonitis	37	36	1	2.7
Diffuse peritonitis	107	67	40	37.28
Local abscess	45	41	4	8.88

In reviewing the etiology I find no cases identified as so-called primary peritonitis of the pneumococcus or streptococcus type, although according to published reports[1, 2] such cases are not rare following respiratory infections, scarlet fever and measles. Bren-

neman[3] has observed a case of fatal peritonitis following rupture of a suppurating mesenteric lymph node. We have seen numerous cases of acute mesenteric lymphadenitis[4] occurring in children and young adults, but so far have seen no peritonitis resulting.

Treatment. The proper treatment of acute local and early spreading peritonitis is with few exceptions prompt operation for relief of the cause. This is generally accepted and requires no comment. If we could time our operative treatment as we desire, we would do all of these operations in the early stage of the disease. We get many patients early and operate with excellent results. However, in spite of the earnest attempts of the profession to educate the public, the patient too often manages the treatment himself during the early stages, and accelerates disaster by taking among other remedies repeated doses of cathartics.

The treatment of acute diffuse peritonitis is a surgical problem; with few exceptions it will involve operation. However, operation is often not the patient's first and most urgent requirement. Treatment may be divided into preoperative, operative and postoperative phases.

Preoperative Treatment. Not infrequently we see brought to the hospital after three or more days of illness a patient who presents a picture of markedly disordered physiology. He is dehydrated from vomiting and inability to assimilate his attempted fluid intake. The absorbing portion of his intestinal tract is paralyzed. Fluid which should pass into the lower portion of the small intestine and colon for absorption is being regurgitated into the upper gastrointestinal tract, where the upper mucosal, biliary and pancreatic secretions are added to it and the mixture is spilling out of the overdistended stomach. The loss of this fluid involves also the loss of other essential elements, particularly chlorides[5]. The patient has been in pain and has been having fever. His reserve energy is depleted. The intestinal wall is infiltrated with toxic inflammatory products as the infection extends to the subperitoneal tissues, and the pressure within the lumen of the gut impairs the intestinal circulation of blood. This, together with a disordered intestinal nerve supply, results in loss of tone. As the abdominal distention increases the patient is further handicapped by respiratory difficulty due to pressure on the diaphragm.

A patient in this condition is obviously not a favorable one to withstand the additional strain of an abdominal operation. However, if we bear in mind the principal factors responsible for his embarrassment, and then remember and use the physiologic meas-

ures available for combating them, it is usually remarkable how much and how quickly the patient's condition can be improved.

In the first place, relieving pain by the use of morphine not only does no harm, but does good. Alton Ochsner[6] has added experimental evidence to support clinical evidence of the value of morphine in acute peritonitis. He has shown that morphine increases intestinal tone, and better tone results in better circulation and more normal intestinal function.

Second, and of greater importance, is the alteration of the patient's body chemistry, his depletion of water and chlorides, and his lack of fuel for production of energy. Intravenous administration of one of several solutions will combat these deficiencies.

We begin with a solution of 5 per cent glucose and 1 per cent sodium chloride. The slightly hypertonic salt solution supplies the urgent need for chlorides. Later on when there appears to be no need for additional chlorides, normal salt solution can be used instead, and if there should be evidence of chloride retention, 5 per cent glucose solution in water alone may be used. The water, of course, is for the relief of dehydration. Coller[7] emphasizes that there is little danger of giving too much water. Any excess is usually excreted in the urine. Patients with normal kidney function who pass about 1500 c.c. of urine in 24 hours are probably receiving a normal amount of fluids. An output of 1500 c.c. of urine can be taken roughly as a guide to the amount of fluid required by an adult patient. In other words, if his urine output is grossly diminished, his fluid intake is probably inadequate. In severe dehydration the patient may pass no urine, and may be suspected of having uremia. Such an adult can easily use from 4000 c.c. to 6000 c.c. of fluid in the first 24 hours. Subsequently he should receive enough to result in his passing of 1000 to 1500 c.c. or more of urine each 24 hours. The glucose received along with the water supplies energy and combats ketosis. Usually the patient can utilize the glucose without the aid of insulin. If sugar appears in the urine in amounts greater than a small fraction of 1 per cent, we give insulin, 20 units to each 40 Gm. of glucose. If the patient is a diabetic the management of that part of his trouble is taken over by the medical and laboratory departments.

The third condition of disordered physiology demanding relief is the over-filled and over-distended small intestine and stomach. Gastric lavage was recommended and widely used for this purpose in the early days of rational treatment of peritonitis. The stomach tube was useful, but as used was comparatively inefficient and its use was distressing to the patient. In 1924, Matas[8] called attention

to his use of the indwelling gastro-duodenal tube (Jutte tube), introduced into the stomach through the nose, and left in place for continued gastric drainage and irrigation. Since that time we have used Matas' method as an adjunct to treatment in all cases of obstructive and paralytic ileus, and I think that the method has saved many lives. Of course gastric drainage is an adjunct to surgical treatment and does not replace operation and should not unduly delay it. The benefit derived from removal of large quantities of foul intestinal contents is apparently not, as was formerly believed, the result of ridding the body of so much toxic material. Wangensteen[9] has shown that while the content of the obstructed intestine is toxic, it is no more toxic than that of normal intestine. The benefit is probably derived from the relief of overdistention and avoidance of the damaging effects which overdistention is capable of producing.

After instituting the above measures, if the patient is not to be operated upon promptly, he is put in a comfortable position in a Gatch bed and a heat tent applied to the abdomen.

Operative Treatment. When we speak of operative treatment of acute diffuse peritonitis, it implies that we have already missed the opportune and safe time to operate for the relief of the condition causing the peritonitis.

Confronted with this situation I would say that operation for acute peritonitis may be divided according to objective into three types:

1. Immediate operation for relief of the causative condition, even if the opportunity has come late.

2. Operations aimed at affecting the peritonitis directly, or combating some immediately harmful process resulting actively from the peritoneal inflammation.

3. Operations for relief of residual pathologic conditions remaining after the acute diffuse process has subsided.

Consideration of the first of these groups brings up the old controversial question of whether or not immediate operation should be done at all in the presence of acute diffuse peritonitis. Undoubtedly the majority of American surgeons have been following the plan advocated by Murphy, and operating promptly for the relief of the condition causing the peritonitis. A smaller group of surgeons, especially when dealing with peritonitis from perforative appendicitis, favor the Ochsner plan of treatment[10, 11, 22]. Mortality rates reported following the use of the Ochsner treatment are usually lower than those reported following immediate operation.

I doubt that we can adopt either method to the extent of entirely excluding the other. Jones and McClure[12] state that Ochsner in a personal communication to them did not claim to use this treatment in all cases of acute general peritonitis, but reserved it for a rather small group of patients that seemed to have little chance of surviving immediate operation. We routinely delay operation until the patients have had time to show improvement from the use of the preoperative treatment already described. However, a majority of our patients are considered operable and the operations are done within six hours after admission. If the patient's condition under treatment failed to improve sufficiently, and if he appeared to have little or no chance of surviving prompt operation, it is delayed and the preoperative treatment continued.

The second type of operative procedure, aimed at directly affecting the peritonitis or some of its active results, includes few if any accepted procedures. One of the first of these was to irrigate the peritoneal cavity, washing out the pus with water or saline solution[13]. This has been abandoned. Attempts to drain the general peritoneal cavity have also been tried and abandoned[14]. Of course, this does not mean that necrotic or sloughing areas at the sight of the causative lesion should not be adequately drained. Various therapuetic substances have been poured into the cavity. Among such substances are ether[15], alcohol [16], acid and pepsin[17], and immune serum[18]. I have had no experience with any of them. Enterostomy has been used to drain the distended intestine. During the twelve years that we have been using gastric drainage by the Matas method we have found enterostomy practically unnecessary. Lymphaticostomy, with the idea of draining away the toxic contents of the thoracic duct has been suggested[19]. I know of no great success of this method.

The third type of procedure for relief of residual conditions when acute peritonitis has subsided, consists mostly of incision and drainage of localized collections of pus. If the abscess is single and small the outlook is good and the operation can usually be safely delayed if desired. Many small abscesses disappear without operation. Patients with large abscesses are in a more serious condition. They may become rapidly septic and they have a tendency to develop complications. Intraperitoneal rupture of the abscess may also occur. Large abscesses should be opened without delay, just as soon as they can be detected. An abscess located in the pelvis may be drained through the rectum or the vagina. When a diffuse peritonitis is followed by the formation of multiple abscesses of appreciable size, in my experience the prognosis is extremely bad.

Before leaving the subject of operative treatment I would like to mention just a few points about operative procedure. Undoubtedly rapid operation is an advantage; but I do not think that gentleness and accuracy should be sacrificed for the sake of speed. The question of how much or how little to do after entering the abdomen is occasionally a very delicate one. We try to do as little as possible and yet if we fail to do what is necessary nothing has been gained by opening the abdomen. In dealing with the appendix I have almost invariably removed it except in cases of delayed operation when it was a part of the wall of a localized abscess and could not be delivered without breaking up the abscess wall. When operating on cases of free peritonitis of appendiceal origin, and when sloughing tissue has to be left in the abdomen, I consider adequate drainage of the greatest importance. In such cases we are using the quarantine drain described by Coffey[20]. In dealing with peritonitis associated with mechanical obstruction of the small intestine I do not agree with those who advocate opening the abdomen, tapping the proximal gut and closing without further procedure. I prefer to locate and inspect the obstructive lesion. If resection is necessary I do not do a primary anastomosis in the presence of peritonitis, but deliver the portion of intestine to be resected through the wound and close around it according to the Mikulicz technic of resection. The small intestinal contents is quite irritating to the skin, but we have controlled this by using a paste of kaolin or of zinc oxide. The spur can be cut early and it is remarkable how soon the intestinal stream is re-established. I have used this procedure three times in this series and rather luckily all three patients recovered. In no instance has the intestine been opened for the purpose of emptying its content by stripping. On the other hand the intestines have been manipulated as little as possible. In dealing with cases of perforated ulcer, I have aspirated the free fluid, repaired the ulcer and drained the abdominal wall. If the tissue surrounding the perforation was too indurated and friable to permit suturing, I have removed enough of it to make suturing possible.

Postoperative Treatment. The treatment after operation is similar to the preoperative treatment already described. In addition during the past two years we have occasionally used hypertonic salt solution to increase intestinal tone in paralytic ileus[21]. The dose has been 20 to 30 c.c. of 20 per cent solution for an adult patient. In my experience this is a useful measure.

SUMMARY

Five hundred ninety-seven cases of peritonitis are reviewed.

They are classified as to cause variety, and mortality.

The treatment used is discussed, with added remarks concerning some therapeutic measures used by others.

The Ochsner treatment of diffuse peritonitis is considered valuable and was used in some cases but not routinely in all cases of diffuse peritonitis.

REFERENCES

1. Brenneman, Joseph: Acute Abdominal Conditions in Children, Colorado Med. *32:* 14-23 (Jan.) 1935.
2. Robinowitz, M. A.: Acute Hematogenous Streptococcic Peritonitis, Am. J. M. Sc. *157:* 797, 1919.
3. Brenneman, Joseph: Personal communication.
4. Wilensky, A. O.: Mesenteric Lymphadenitis, Med. Rec. *98:* 770 (Nov. 6) 1920.
5. Haden, R. L., and Orr, T. G.: Upper Intestinal Tract Obstruction, J. Missouri M. A. *20:* 185 (June) 1923.
6. Ochsner, Alton: Postoperative Treatment Based on Physiologic Principles, South. Surgeon *4:* 197 (June) 1935.
7. Coller, F. A.: Water Balance in Surgical Patients, Nebraska M. J. *20:* 365-368 (Oct.) 1935.
8. Matas, Rudolph: The Continuous Intravenous Drip, with Remarks on the Value of Continued Gastric Drainage Irrigation by Nasal Intubation with Gastroduodenal Tube (Jutte) in Surgical Practice, Ann. Surg. *79:* 643 (May) 1924.
9. Wangensteen, O. H., and Chunn, S. S.: Studies in Intestinal Obstruction; Comparison of Toxicity of Normal and Obstructed Intestinal Content, Arch. Surg. *16:* 606-614 (Feb.) 1928.
10. Coller, F. A., and Potter, E. B.: The Treatment of Peritonitis Associated with Appendicitis, J. A. M. A. *103:* 1753 (Dec. 8) 1934.
11. Jopson, J. H., and Pfeiffer, D. B.: The Limitations of the Ochsner Treatment in Certain Types of Peritonitis, Ann. Surg. *77:* 194 (Feb.) 1923.
12. Jones, D. F., and McClure, William L.: Practice of Surgery, in Dean Lewis' Modern Surgery *1:* 29, Hagerstown, Md.: W. F. Prior Co., Inc., 1929.
13. Hartwell, J. A.: The Treatment of Spreading Peritonitis, Ann. Surg. *53:* 146, 1911.
14. Blake, J. A.: Drainage, Ann. Surg. *75:* 385 (April) 1922.
15. Souligoux and others, quoted in Foreign Letters, J. A. M. A. *88:* 1192 (April 9) 1927.
16. Behan, R. J.: Treatment of Localized and Acute General Peritonitis by Intraperitoneal Lavage with Alcohol, Tr. Am. Therap. Soc. *33:* 30-40, 1933.
17. Schoenbauer, L.: Clinical and Experimental Studies of Serous Cavities, Archiv f. Klin. Chir. *140:* 1-8, 1926.
18. Gundel, M., and Suesbrich, F.: Die Serumbehandlung der Peritonitis and ihre wissenshaftlichen Grundlagen, Zentralbl. f. Chir. *61:* 306-325 (Feb 10) 1934.
19. Costain, W. A.: Lymphatic Drainage, New York State J. Med. *26:* 225-231 (March 15) 1926.
20. Coffey, R. C.: Application of Quarantine Drainage in Abdominal Surgery, Am. J. Surg. *24:* 417 (May) 1934.
21. Usadel: Abstract., Progressive Med. Page 81 (June) 1928.
22. Ochsner, Alton: The Conservative Treatment of Appendiceal Peritonitis, New Orleans M. & S. J. *87:* 32-38 (July) 1934.

SURGERY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

CLEVELAND D. WHELCHEL, M. D., F. A. C. S.
Gainesville, Georgia

There is not time to go into the history of pulmonary collapse therapy and give due credit to the pioneers who blazed the trail that we are following. However, I should like to mention James Carson, who in 1820 suggested the use of artificial pneumothorax, Carlo Forlanini who first performed this operation in 1888, de Cerenville who attempted the first thoracoplasty in 1885, Tuffier who performed the first extrapleural pneumolysis in 1891, and Stuertz who recommended simple section of the phrenic nerve in 1911.

We have no record of the first pulmonary collapse as nature did this with the first pleural effusion. It is stated that 95 per cent of all idiopathic pleurisies with effusion are tuberculous and the remaining 5 per cent should be considered so until proven otherwise. How many of us have withdrawn clear straw-colored fluid from the pleural cavity in these cases, more than enough to relieve the respiratory embarrassment, only to allow the lung to reinflate thereby defeating nature's method of treating a diseased lung. Today following nature's lead we have improved upon her method of collapse, so that now a choice of method is available which will be suitable in almost any case. Many times it becomes advisable to combine two or more of these procedures in order to accomplish the desired result.

Surgery is in the same position in the treatment of pulmonary tuberculosis today that it was in the treatment of acute abdominal disease twenty-five years ago. There are some who still believe that bed rest alone if persisted in will cure most of the cases. This has not been our experience. We feel that every patient should be studied to see if collapse therapy is advisable. How many physicians of today when called to see a case of acute appendicitis would treat this patient with morphine and purgatives, hoping for a favorable outcome? This method of treatment lost its advocates, first when the physician lost his patient, second his courage, and third his conviction. I believe the same holds true today in the treatment of pulmonary tuberculosis. If suitable collapse measures are insti-

From the Georgia State Sanatorium for Tuberculosis at Alto.
Read at Northeast Georgia Sectional Meeting of The Southeastern Surgical Congress, Royston, Ga., on Oct. 16, 1935.

tuted in the early stages of the disease the far-advanced cases will be cut to a minimum.

Obviously in a paper of this character it will be possible to discuss only the more important methods of collapse therapy and these in a very brief manner. Before the development of collapse therapy only the occasional patient developed a cure. Since these various procedures have been instituted the average case has certainly now 75 per cent better chance than before.

Of the various procedures used to promote collapse, artificial pneumothorax is the method of choice in most cases. It will accomplish an arrest or cure in a large number of patients if it can be properly instituted. Unfortunately, however, in about 50 per cent of the cases this is not possible because of the presence of adhesions. Unfortunately also these adhesions are invariably present over the cavities, the very part of the lung which is in greatest need of compression or collapse.

However, in many of these cases the offending adhesions may be severed by means of closed intrapleural pneumolysis and the unsatisfactory pneumothorax be converted into a successful one. Before performing this procedure a stereoscopic film study is essential in order to determine the best approach, what adhesions must be cut, etc. It must be remembered, however, that only the thoracoscopic examination is conclusive. I can recall several cases in which perfect closure of cavities was accomplished by severing adhesions to the posterior chest wall, none of which were visible on the x-ray film. On other occasions adhesions which seemed inoperable on the x-ray film were found to be operable on thoracoscopic examination. Even in those cases in which there is not an operable distance between the cavity and the chest wall it is sometimes possible to coagulate the adhesion at its parietal attachment. This often results in the adhesion relaxing and degenerating to such an extent that the cavity can close without its complete severance. Obviously the safe performance of this procedure requires thorough orientation regarding the thoracoscopic appearance of the pleural cavity, both in its normal state and as altered by pneumothorax treatment and disease.

Some authors have advocated opening the pleural cavity in order to sever the adhesions. However, in my hands the closed intrapleural method has been so efficient that I have never seen the necessity of this more hazardous open method.

Although pneumothorax is usually the procedure of choice, in those cases in which pleural adhesions make it impossible or incomplete, an associated phrenic nerve operation is often of great value.

By paralyzing the corresponding half of the diaphragm the thoracic cage is diminished in size and the lung tissue relaxed. This allows further compression and immobilization of the tuberculous lung and also releases the pull of adhesions upon any cavities that might be present thus allowing them to close. It is not uncommon to see cavities which had been open for a period of months to close rapidly following this operation.

The proper use of the phrenic nerve operation requires judgment and careful individualization in each case. Such factors as whether the disease is unilateral or bilateral, apical or basal, whether the operation is likely to spread the disease to the good side, whether the procedure is to be used in conjunction with a pneumothorax in the same or opposite side, etc., determine on which side the operation should be performed, and whether a temporary or permanent interruption should be made.

In a certain number of cases a satisfactory collapse of the diseased lung cannot be obtained by any of the methods that we have just considered. Such patients are candidates for extrapleural thoracoplasty. This procedure, although a valuable one, because of its inherent dangers must be used with a fine discrimination. It certainly should not be considered an operation of last resort but rather should be confined to good operative risks. Briefly, such patients should be under 50 years of age, should show x-ray evidence of fibrosis and mediastinal fixation, clinical evidence of unilateral relatively inactive disease, and no evidence of progressive activity or any complicating extrapulmonary tuberculous lesions.

I feel that it is advisable to precede complete thoracoplasties with phrenic exeresis. Also that the operation unless purely an apical one should be done in three or four stages. These stages should be performed as close together as safety permits so that there will not be time for the periosteum to form new bone, as this increases the technical difficulties of the operation. The ribs which are resected subperiosteally should be removed right up to the corresponding transverse processes in order not to leave a gutter along the side of the spine. Different operators use different incisions and remove various length segments of ribs. I prefer the J incision and in the average case try to remove about 2½ to 3 inches of the first rib, 4 to 5 inches of the second, 6 to 7 of the third, and 7 to 8 inches of the fourth rib. In removing the ribs care must be exercised not to tear into the lung as tuberculous empyema and wound infection may follow such an accident. Since the operation often causes considerable shock I try not to keep the patient on the table longer

than 30 or 40 minutes. However, thoroughness must not be sacrificed for speed. Drainage is instituted for from 24 to 48 hours.

If the patient is coughing up much sputum or has a cavity with a fluid level, postural drainage is performed immediately before the operation. It is sometimes advisable to have the head somewhat lowered during the operation to facilitate drainage from the tracheobronchial tree and prevent its aspiration into the good lung. Nitrous oxide-oxygen anesthesia is used. The patient is usually matched for a transfusion. If a large pneumothorax pocket is present it is either emptied of air during the operation or the day before.

There is a group of cases of unilateral or bilateral apical cavitation in which pneumothorax is not possible and thoracoplasties either inadvisable or ineffective. These may often be successfully handled by a procedure known as plombage. This consists of collapsing the cavities by packing paraffin between the overlying parietal pleura and the chest wall. The procedure while of distinct value has certain inherent dangers and is limited to those cases in which the pleural space is obliterated and the cavities being compressed have thick enough walls to prevent the paraffin from rupturing into them.

In conclusion, surgery in the treatment of pulmonary tuberculosis endeavors to accomplish rest, immobilization, and compression by collapsing the diseased lung by one method or another. In fact we feel that collapse therapy is so valuable that at our institution we use it in one form or another in 80 per cent of all cases, and in over 90 per cent of all good cases.

Therefore we may say that:

1. Of the various forms of collapse therapy artificial pneumothorax, if successful, is the method of choice. This procedure is indicated in from 80 to 90 per cent of all cases of pulmonary tuberculosis. However, in less than half of these cases is it possible to perform a satisfactory pneumothorax. When a satisfactory collapse can be obtained 60 to 70 per cent of the patients will become clinically well or arrested. When the collapse is unsatisfactory only about 25 per cent will become clinically well or arrested.

2. An unsatisfactory pneumothorax after a four to six month trial should be converted into a satisfactory one by closed intrapleural pneumolysis. This procedure will be successful generally in 70 per cent of the cases. However, in my series of cases about 85 per cent have resulted favorably. In my series of closed intrapleural pneumolysis there has not been a single death.

3. Phrenic nerve operation is of distinct value. An analysis of the results obtained is being made at the present time and will be reported shortly. However, in our series of over 600 phrenic operations we have had only one death—the patient died 24 hours later due to respiratory embarrassment.

4. In selected cases in which pneumothorax is not possible or effective extrapleural thoracoplasty is the procedure of choice under optimum conditions. From 60 to 75 per cent of the patients should show definite improvement. The operative mortality should not be over 10 per cent.

5. In certain groups of cases with apical cavitation in which other methods of collapse therapy are not effective plombage will be of value. A high percentage of closure of cavities will occur. The final mortality should be less than 3 per cent.

6. Finally it must be thoroughly understood that all collapse measures are to be undertaken only in conjunction with bed rest, and as near complete rest of mind and body as is possible to obtain. If collapse methods are undertaken early I believe the convalescent period will be spoken of as a period of months rather than as heretofore a period of years.

NEOPLASMS OF THE RECTUM

Incidence, Inter-relationship and Diagnostic Criteria.

CURTICE ROSSER, M. D., F. A. C. S.
Professor of Proctology
Baylor University, Department of Medicine
Dallas

AN awakened interest in proctologic problems, the more general employment of the proctoscope and continually increasingly effective roentgenologic methods for visualizing the interior of the large bowel have brought into notice the unusual variety of tumors which may be found in this location. Neoplasms such as fibroma, lipoma, myoma, angioma, endothelioma, dermoid and lymphoma are being reported as occasional intrarectal discoveries and while the group just enumerated are too rare in incidence to merit detailed consideration in a brief review the possibility of their presence requires their inclusion in differential diagnosis.

For practical purposes the benign tumors which by reason of frequency of occurrence, diagnostic differentiation or possibility of malignant degeneration are of constant importance are adenomas, adenomyomas and eleomas. The adenoma is by far the most common. Dewes[1] in a review of 101 cases of benign rectal tumors reports that 81 were adenomas. Buie[2] found that 2.5 per cent of all persons proctoscoped at the Mayo Clinic over a period of four years had one or more polyps. The foreign body granuloma, eleoma, which is produced by the injection of oil in rectal tissues in the treatment of hemorrhoids is undoubtedly of high incidence in all communities in which this form of therapy is popular, and in my own observation has been the most commonly encountered benign tumefaction. Endometriosis involving the rectosigmoid and rectum is either increasing or is being recognized more universally.

Adenomas are encountered as polyps, villous papillomas or in the condition known as multiple polyposis or adenomatosis. The typical adenomatous polyp of the rectum is first seen as a small rose-hued submucous swelling which gradually increases in size and becomes polypoid by developing a pedicle of mucous membrane. Approximately 50 per cent of these tumors indicate their presence by bleeding, discharges of pus or mucus, local irritation or protrusion on defecation. While their nature is evident upon direct visualization, the ever-present possibility of malignant degeneration

Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

compels their removal and sectioning as a routine diagnostic procedure. Rankin and FitzGibbon[3] have reported 13 cases in which the change from adenoma to adenocarcinoma was clearly demonstrated. Dukes[4] reported the finding of associated adenomas in 75 per cent of 33 consecutive operative specimens of cancer of the rectum and traced clearly the pre-malignant changes in this type of tumor. Removal of such polyps is simplified by employment of the snare and coagulating current.

Villous papilloma is a slowly growing, soft, fleshy tumor with a large base which in advanced cases will be found to cover a considerable area of the rectal wall. Diarrhea and the constant discharge of very large amounts of clear mucus are the two usual

Fig. 1. Adenomyoma invading the wall of rectum. a. Mucosa of bowel. b. Isolated deposit of endometrial tissue.

symptoms. Bensaude[5] estimates that malignant change supervenes in 45 per cent of these papillomas although many will be present for years before this change takes place. Rectal resection however is often indicated for large growths even though biopsy is negative.

Multiple adenomatosis when not found in association with tuberculosis or ulcerative colitis, is now believed to be a familial disease. Malignant transformation is conceded to occur eventually in practically all cases. Doering [6] found that in 50 cases reviewed by him there had been 31 deaths from carcinoma. As seen through the proctoscope these tumors will be found to be both sessile and pedunculated, varying considerably in size. The rectum and sigmoid are chiefly involved. Symptoms are always present, consisting of cramping, pain, diarrhea and appearance of bloody mucus. In spite of

more encouraging recent reports concerning complete colectomy my own preference favors the conservative management of these cases, including snare and coagulation removal of those adenomas which can be reached through the proctoscope, bland diet and, where diarrhea and colic are troublesome, appendicostomy to permit irrigations to clear the bowel of blood, mucus and toxic products. Routine re-examinations are indicated and when and if malignancy occurs, usually in the rectum or sigmoid, at that time the proper portion of bowel may be radically excised.

It is a fairly common finding to note a small single adenomyoma in the rectovaginal septum in the course of a routine proctoscopic examination on a female. Occasionally the patient gives a history of pain during the menstrual period referred to this locality but as a general rule small tumors of this type are symptomless. Larger masses have been observed to infiltrate the sigmoid and rectum and these give rise to serious complications. The usual history in such a case describes a female of 30 to 45 years of age who has not recently given birth to a child and whose chief complaints are gradually increasing constipation and a dull pain in the lower left side of the abdomen which is increased during the menstrual period. Mucus and blood are late or absent, as erosion through the intestinal mucous membrane seldom occurs.

The condition may be found as either single or multiple tumors invading the muscular and submucosal layers of the lower sigmoid and upper rectum. Differential diagnosis from sarcoma of the rectum and from some of the less common benign tumors is not difficult if the growth is small and found only in the rectovaginal septum, but may be quite confusing if the process has invaded the walls of the rectum or sigmoid at some distance from its origin (Fig. 1). Sarcoma, for example, is frequently a smooth elastic tumor in the muscular coat of the bowel and erodes through the mucous membrane producing superficial ulceration quite late in its course. Of six cases of sarcoma reported by Smith[7] in 1933, in one there were numerous submucous nodules, in another a massive edema of the mucosa, in one sessile polyps and in the remainder an ulcerated surface had developed. Kallet[8] in 1932 reported seven cases of sarcoma of the rectum. In six of them the growths were on the anterior wall. The first objective manifestation in four of these cases was the presence of a mass beneath the mucosa and the long period during which the mucosa remains intact, unlike adenocarcinoma where ulceration and appearance of blood in the stool occurs early, was commented upon by Kallet. With these facts in mind, it must be conceded that biopsy will occasionally be necessary to conclude the differential diagnosis.

Some years ago the presence of firm, non-ulcerated submucosal tumefaction became so common an observation in the proctoscopy of patients previously given injections in a number of cities and villages in Texas that biopsy was performed in 22 cases presenting this phenomenon and it was thus determined that the indurated yellowish lumps were not xanthomatous nor composed of simple fibrous tissue but were foreign-body granulomas from non-absorbed oil. In 1932 Wallace and I[9] reviewed our investigations which included the reproduction of these tumors by experimental injections

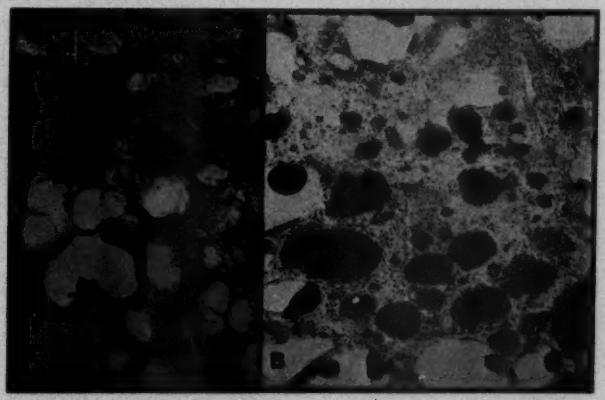


Fig. 2. Eleoma. a. Photomicrograph of section from eleomatous mass, stained with hematoxylin. Apparent vacuoles are seen, surrounded by a thick fibrous stroma. b. Same section, stained with Sudan—III. The apparent vacuoles are shown to contain the injected oil.

of oil into human rectal tissue. We had determined that mineral oil was the prime etiologic agent and that certain vegetable oils in large amounts would also produce this type of tumor. The eleoma grossly is suggestive of sarcoma of the rectum, the chemical stricture which results from coalescing eleomas, when erosion of the mucosa is present, is not unlike annular carcinoma in appearance, and the microscopic sections, which show giant cells, may resemble tuberculosis. Where the diagnosis remains uncertain after proctoscopy of an individual giving the history of submucosal injections the use of a fat stain on a bit of excised tissue defines the picture by revealing the presence of the injected oil in spaces which appear empty when the usual stains are used (Fig. 2).

With the exception of sarcoma which occurs in the rectum in an incidence of 1 to 200 carcinomas, the malignant neoplasms of the rectum are basically epithelomas and adenocarcinomas. Contrary to general medical opinion, it is becoming more and more apparent

that a very large majority of rectal cancers will be found to originate not in the ampulla but in either the narrow rectosigmoid canal by which the bolus enters the rectum or in the constricted anal canal by which it makes its exit. Whether or not this distribution indicates that trauma from constriction and angulation serves as an etiologic agent in the production of rectal malignancy is a debatable point. Certainly it is apparent that if Tuttle, W. J. Mayo and Rankin were correct in their opinion that more than 65 per cent of such cancers are located in the rectosigmoid the majority of rectal cancers in their earliest stages will *not* be detected by a simple digital examination, as tumors in this locality prolapse within reach of the finger only in advanced stages.

Nor is the usual statement that epitheliomas occur only in the anus and adenocarcinomas only above the dentate line borne out by my observations.

In a recent analysis of 25 cases of cancer of the anal canal observed during a 10 year period,[10] I found that the majority of the patients were under 51 years of age; that 84 per cent had some associated benign lesion—fissure, fistula, hemorrhoids, polyp, cryptitis—and that either squamous cancer (which comprises about 3 per cent of all rectal neoplasms) or adenocarcinoma could be present and that either type may be found above or below the dentate line.

Adenocarcinoma, for example, may originate at the opening of a fistulous tract or in one of the vestigial glands which Hermann[11] and Tucker[12] have described, and extend to the perianal epithelium; squamous cancer may extend from its origin on the dentate line to the upper portion of the anal canal, or may originate on squamous-celled metaplasia of the cylindric-celled epithelium above the dentate line consequent to the trauma of prolapsing hemorrhoids (Fig. 3). I have become convinced from the constantly observed association of benign lesions with malignancy in the anal canal that the development of cancer in this locality is in some manner related to the previous existence of such lesions, and have previously reported a group of hemorrhoids and fistulas which we have every reason to believe were primary and in which epitheloma and adenocarcinoma had resulted. Buie and Brust[13] stated that benign lesions were found in intimate association with the neoplasm in 34 of 51 cases of epithelioma of the anus observed by their group, which data they believe support the hypothesis which I first advanced in 1931[14] that previous chronic infections and irritations are a predisposing cause of anal carcinoma, just as the previous existence of benign polyposis or ulceration is a recognized predisposing agent in cancer of the ampulla and rectosigmoid.

In my experience pain was the presenting symptom in 60 per cent of anal cancers; only 6 of the 25 patients studied were constipated; 16 had noted passage of blood, usually a streak on the paper. The average duration of symptoms, in spite of the accessibility of the lesion, exceeded 9 months.

The diagnosis of rectal cancer is therefore complicated by the constant necessity for eliminating the presence of malignant degeneration in ancient anal lesions, in rectal polyps and in the ulcers

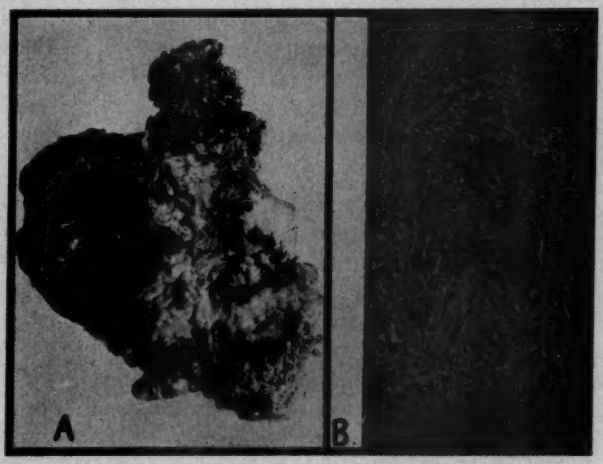


Fig. 3. a. Ancient hemorrhoids with superimposed squamous cancer. b. Photomicrograph of lesion shown in a.

of colitis. The earliest symptoms of anal and rectal carcinoma are concerned with the presence of such predisposing lesions plus certain additional signs, never pathognomonic in themselves, often vague, but sufficient to suggest to the alert physician the advisability of a careful, complete and expert rectal examination.

Probably the most constant early sign is a sense of discomfort in the rectum, which is not relieved by defecation. Bleeding may not be severe and bleeding may arise from other causes; however, all rectal cancers bleed. Pain is usually present early in anal cancer, rarely is this true in cancer of the ampulla and rectosigmoid although referred pain to the sympathetic gangloin of the sacrum or lower abdomen is sometimes observed.

In the intermediate stage of the development of the neoplasm, the usual symptoms are constipation or diarrhea, discharge of blood alone or mixed with mucus or pus, pain (in cancer at or near the anus) and slight weight loss. Alternating diarrhea and constipation

is actually an uncommon symptom and the textbook syndrome of weight loss, cachexia, obstruction and abdominal distention which accompanies final stages are signs too late for hope of favorable outcome.

COMMENT

There is a curious and constant relationship between all the commoner rectal neoplasms; the thread that binds the group together is the ever present specter of malignancy.

The benign adenoma of today may tomorrow contain the nucleus of cancer; invading endometriosis must be differentiated from sarcoma and, where extensive bowel infiltration has occurred, has in itself many of the characteristics of local malignancy; the factitial tumor, eleoma, presents a definitely cancriform picture.

It is apparent that accurate and comprehensive diagnostic measures are essential in the presence or possibility of any rectal neoplasm. The entirely laudable attempt to popularize rectal examinations on the part of the practitioner has at the same time deeply inculcated a peculiar dogma that this procedure may properly consist entirely of the casual introduction of the index finger. The fact that the majority of rectal tumors in their curable stages are in the upper rectal zones suggests that careful direct visualization of the entire rectum and rectosigmoid is also indicated; the fortunate trend of opinion which has rescued biopsy from an undeserved obliquity affords an additional safeguard to the patient where the presence of malignancy or the degree of malignancy present are undetermined.

710 Medical Arts Building.

REFERENCES

1. Dewes, J. W.: Consideration of Various Forms of Benign Intestinal Tumors, Boston M. & S. J. *155:* 427, 1906.
2. Rankin, F. W.; Bargen, J. A., and Buie, X. A.: The Colon, Rectum and Anus. Philadelphia: W. B. Saunders Co., 1932, p. 390.
3. FitzGibbon, G., and Rankin, F. W.: Polyps of the Large Intestine, Surg., Gynec. & Obst. *52:* 1136 (June) 1931.
4. Dukes, Cuthbert: Simple Tumors of the Large Intestine and their Relation to Cancer, Brit. J. Surg. *13:* 720 (Apr. 26) 1926.
5. Bensaude, R.; Cain, A., and Lambling, A.: Villous Tumors, Presse Med. *101:* 1713, 1930.
6. Doering, Hans: Die Polyposis Intestine, Arch f. klin. Chir. *83:* 194, 1907.
7. Smith, N. D.: Lymphosarcoma of the Rectum and Sigmoid, Tr. Am. Proct. Soc., *32:* 160, 1933; also Proc. Staff Meet., Mayo Clin. *8:* 437-438 (July 19) 1933.
8. Kallet, H. I., and Saltzstein, H. C.: Sarcoma, Melanoma and Leukosarcoma of the Rectum, Tr. Am. Proct. Soc. *33:* p. 75, 1932.

9. Rosser, Curtice, and Wallace, S. A.: Tumor Formation; Pathologic Changes Consequent to Injection of Oils under Rectal Mucosa, J. A. M. A. *99:* 2167 (Dec. 24) 1932.

10. Rosser, Curtice: Cancer of the Anal Canal (Survey of 25 cases), South. M. J. *28:* 527 (June) 1935.

11. Herrmann, Gustave (1880) quoted by Pennington, Diseases of Rectum. Philadelphia: P. Blakiston's Sons & Co., 1923.

12. Tucker, C. C., and Hellwig, C. A.: Histopathology of Anal Crypts, Surg., Gynec. & Obst. *58:* 145 (Feb.) 1934.

13. Buie, L. A., and Brust, J. C. M.: Malignant Anal Lesions of Epithelial Origin, Lancet, *53:* 565 (Nov. 1) 1933.

14. Rosser, Curtice: Etiology of Anal Cancer, Am. J. Surg. *11:* 328 (Feb.) 1931.

THE THERAPEUSIS OF LYMPHOGRANULOMA INGUINALE

EMMERICH VON HAAM, M. D.
Assistant Professor of Bacteriology and Pathology,
Louisiana State University Medical Center
New Orleans

Nothing better characterizes our present knowledge of the therapeusis of lymphogranuloma inguinale than the words with which the great continental expert in this disease, Sven Hellerstroem, introduced his answer to a questionnaire on this subject for a dermatologic journal: "It is a problem which at present is far from being solved even in the hands of the trained specialists." Nevertheless, the physician consulting the current literature will find an amazing amount of matter recommending apparently successful treatment of the disease by widely different methods. To the critical reader of these reports, however, three facts will be apparent: first, that many authors are unaware of the true character of the disease; second, that comparative investigations of various therapeutic methods by the same authors are rare; third, that in most instances the number of cases reported treated is very small. Our need for systematic therapeutic research, however, is clearly demonstrated by the increasingly wide distribution of the disease whose recognition has been so facilitated by the introduction of Frei's specific intracutaneous reaction.

According to our present knowledge, lymphogranuloma inguinale is caused by a filtrable virus which enters the human body most frequently through the skin or the mucous membranes of the genital organs producing a characteristic inflammatory process followed by suppuration and extensive sclerosing fibrosis of the deep and superficial regional lymph glands. On the basis of clinical and experimental observations we may rightly assume that a passing general infection of the organism with the virus is quite frequent and leads to constitutional symptoms and under certain circumstances produces distant lesions in the joints, the skin, the eyes and other organs[7, 10]. Months or years after the acute symptoms of the disease have subsided, elephantiasic enlargement of parts of the external genitalia (labia, scrotum), sometimes with extensive ulceration, is observed in a certain number of cases fol-

From the Departments of Pathology and Bacteriology of the Medical Center of Louisiana State University and the Charity Hospital of Louisiana at New Orleans.
Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

lowed frequently, especially in the female, by inflammatory stricture of the rectum and severe proctitis. This is thought by the majority of authors to be a consequence of the destroyed lymph drainage[20]; some, however, regarding it as a manifestation of the action of an active virus which can be demonstrated by animal experiments still in the diseased tissues[7]. While the disease rarely leads to the death of the afflicted individual, it brings much discomfort to its victims, many of whom are for months or years unable to do any work and fill the charity clinics of our welfare institutions. Since our methods of preventive medicine and public hygiene are still powerless to prevent infection, it appears logical that we should exert every effort in attempting successfully to combat the disease when once established.

We have studied rather extensively during the past years the virus of lymphogranuloma inguinale[3] and were struck by the great incidence of this infection amongst the colored population of New Orleans. This offered splendid opportunity for a systematic investigation of the comparative value of the various therapeutic methods recommended. These methods can be divided in three general groups[22]: methods of physical, medical and surgical therapy. Gay-Prieto[8] emphasizes that there is no treatment that is equally successful for all the manifestations or types of lymphogranuloma inguinale, and Rousseau[25] and his co-workers also warn against adopting any one form of therapy. In the following sections I will discuss separately the therapeutic methods used in the treatment of the early and late or secondary manifestations of lymphogranuloma inguinale.

Treatment of Acute Lymphogranuloma Inguinale

The primary lesions of this infection, which may appear in the form of a small herpetiform lesion, a mild nonspecific urethritis or in rare instances resemble a phagedenic ulcer, have great tendency to spontaneous healing and do not require special therapeutic attention. In most cases the lesion is so minute that it is completely overlooked by the patient and no evidences of it can be found at a later examination. The severe constitutional symptoms which are usually present at the onset of the disease can be combated effectually by such general therapeutic measures as rest in bed, light diet and purgation. In many cases we observed the favorable action of salicylates, especially aspirin. Quinine, which had been used extensively before the true character of the disease was known[16], has no place in the therapy of lymphogranuloma inguinale. Most therapeutic efforts have been centered upon the specific regional lymphadeni-

tis which represents the principal manifestations of acute lymphogranuloma inguinale. Chevallier and Bernard[4] have differentiated five steps in the clinical evolution of these buboes, each being characterized by a different pathologic picture. Simple, firm induration of the gland produced by massive hyperplasia of the reticulum is followed by periadenitis with fixation of the glands in an inflammatory mass. Later the appearance of suppuration is evidenced by distinct fluctuation of the enlarged and adherent glandular masses, this finally leading to the formation of numerous fistulas, the surface of the lesion then "resembling the mouthpiece of a watering pot." The healing of this fistulous mass is slow and characterized by a sclerosing process which will replace the destroyed glandular tissue by fibrous tissue, thus severing lymph drainage. In addition to this usual clinical picture, the evolution and healing of which requires a period of from several months to several years, abortive forms may be observed as well as types with massive suppuration probably due to virulent secondary pyogenic infection. The evaluation of the effective therapeutic methods in the treatment of lymphogranulomatous adenitis is, as Stannus[30] correctly emphasizes, greatly handicapped by the fact that the abortive forms occur with much more frequency than has been previously assumed. This may also be the reason why early treatment of the climatic bubo, which includes all the abortive forms, is more successful[11, 12] than is treatment after fluctuation has been observed.

The physical methods of therapy employed in the treatment of the acute bubo of lymphogranuloma inguinale comprise chiefly the use of galvanic cauterization[32], ultraviolet rays, x-rays [28, 18] and radium. None of those methods are credited with outstanding success and should be applied only in combination with medical or surgical treatment.

The medical therapy consists of the use of drugs or biologic products such as specific and nonspecific vaccines and proteins. Amongst the drugs the group of antimony preparations has been used most widely in the therapy of the acute bubo and has in the hands of some investigators proven a rather successful remedy. Tartar emetic and various commercial preparations (stibenyl, neostibosan, anthiomalin and fuadin) are used intramuscularly or intravenously and healing, sometimes without suppuration, is reported in from four to sixteen weeks following their use. Strong drug reaction is desirable[29], and nausea, muscular pains and occasionally severe albuminuria have been produced by rapid injection of the drug. Sezary and Lenégre[28] doubt on the basis of their experience the therapeutic value of the antimony preparations. Our own limited

experience with tartar emetic confirms the opinion of the French authors to such an extent that we have become accustomed to use the failure of this type of therapy as a differential diagnostic characteristic between the chronic lesions of lymphogranuloma inguinale and similar lesions caused by granuloma inguinale for which tartar emetic is a specific drug. The numerous other remedies recommended by various authors lack sufficient investigation to establish their therapeutic value. Iodides, methylene blue, various copper salts, psorimangan, introcid, sodium salicylate and gold salts, in the form of solganol, have been tried in a small number of cases with varying results. As most harmless of those mentioned drugs, sodium salicylate has recently found wide use, especially by French dermatologists. It is recommended[5] in oral doses of 6 to 8 Gm. a day accompanied by a strict milk diet in order to avoid gastric complications or in the form of intravenous injections of sodium salicylic gluconate. Aspirin given in small doses has with us proven an excellent agent in combating the severe constitutional symptoms usually present during the onset of the disease. With increasing frequency attempts to treat lymphogranuloma inguinale with specific or nonspecific vaccines or proteins are reported. The fact that they are usually less harmful than the heavy metals recommends at least their trial although the number of reported "cures" is at present not sufficient to warrant any conclusions. Injections of milk, TAB vaccine, tuberculin or pyrifer apparently have a tendency to hasten the maturation of the glands, while specific vaccines prepared from the pus or gland emulsions from humans afflicted with the disease or obtained from animal organs experimentally inoculated with the virus are supposed to bring about resolution of the process without suppuration. Hellerstroem[11, 12], who instigated the specific vaccine therapy in lymphogranuloma inguinale, recommends a slow desensitization of the allergic patient by small repeated intravenous injections of Frei's antigen[6], and Gay-Prieto[8], based upon the experience of only three cases, looks upon this treatment as the method of choice. Ravaut, Levaditi and Maisler[24] observed healing of the lesions in six cases after one to five intravenous injections of from 0.2 to 0.5 c. c. of antigen. Wien and Perlstein[33] report marked improvement of sixteen cases after repeated intradermal injections of antigen and we have repeatedly observed marked subjective improvement of the patient even after a single diagnostic intradermal injection. Kalz and Sagher[14] report in thirty cases treated by intramuscular injections of convalescent serum healing of the lesions in about six weeks and Tamura[31] recommends treatment of acute bubos with specific anti-lymphogranuloma goat serum which produced cure in his three cases after an average duration of eight weeks.

Surgical treatment during the acute stage of the infection must still be regarded as the most popular method of therapy although it has recently been severely criticized by those who regard lymphogranuloma inguinale as a systemic infection. Simple aspiration of the fluctuating glandular masses with or without subsequent injection of antiseptic solutions, broad incisions with drainage and more or less extensive extirpation of the enlarged glands have been used long before the causal agent of the disease was known. That surgical therapy still holds its place in the treatment of acute lymphogranuloma inguinale can be seen from the fact that in many of the recent publications advocating differing methods of treatment reference is still made to surgery as a last resource in obstinate cases. Slow healing wounds and postoperative elephantiasis of the genitalia as a consequence of the destruction of the lymph drainage are emphasized as undesirable end results of surgery by its opponents[17, 28], while those in favor of this means of therapy stress the great acceleration of complete healing (three to six weeks), with immediate relief of local pain as great advantages of the operative treatment[25, 26]. While we with others[7, 10, 30], have shown that general infection during the course of lymphogranuloma inguinale actually occurs in a rather large number of cases, we also noted that in the majority of patients only local lesions can be observed, due to the rapid destruction of the virus in the organism. Removal of those local lesions therefore would remove the main source of further spread of the infection and is a biologically correct method of treatment. This fact will gain in importance if it is ever proven that the virus of lymphogranuloma inguinale like other true pathogenic viruses multiplies only intracellularly. Another possible advantage of surgical therapy was pointed out by Jersield, who observed that partial adenectomy or only simple incision of a superficial bubo results in marked improvement of the deeper seated lymph glands. He explained this observation which was confirmed by Hellerstroem[11, 12], Rousseau[25] and others on the basis of the effect of autovaccination with antigen through the operative wound. The necessity of surgical intervention in cases of secondary infection with massive suppuration is naturally generally admitted.

Because of those strong and vastly different arguments for and against the value of surgical therapy in acute lymphogranuloma inguinale we decided to reinvestigate the problem. For this purpose, in a series of selected cases with clinical and immunologic diagnosis of the disease, operative procedures were recommended as the proper therapeutic measure and the patients were carefully observed as regards healing of the lesion and general improvement. In order to avoid the abortive form, which will heal under any

therapy, we did not follow Hellerstroem's advice of early operation, but treated all patients conservatively until the size and the consistency of the glands made any spontaneous regression of the lesion appear extremely improbable. A brief survey of the series so treated is given in Table I.

TABLE I

Type of Treatment:	*Number of cases discharged as cured after:*														*Average period of disease: weeks:*
	1	2	3	4	5	6	7	8	9	10	11	12	*4—12 months*	*Over one year*	
						weeks									
Group A: 43 cases															
Partial Adenectomy	1	2	6	14	4	4	4	4	1	.	.	1	2	.	4.7
Group B: 42 cases															
Incision and drainage	.	.	6	8	4	10	4	4	.	.	2	.	2	2	5.0
Group C: 19 cases															
Abortive forms															
(No special treatment)	.	1	1	3	2	4	1	2	.	2	.	2	1	.	6.2

Partial adenectomy resulted in healing of the lesions one month after operation in over 50 per cent of the cases. Simple incision with drainage delayed the healing process in the majority of cases for from two to four weeks. In group C we have listed 19 abortive cases of lymphogranuloma inguinale which came under our observation during the period of the present investigation. These represent the mildest form of the infection without suppuration. Disappearance of the glands or spontaneous cure was observed in the majority of these cases after four to six weeks—equally as long a period as was required for healing of the acute suppurating bubo after surgical therapy. The average period of the disease was 4.7 weeks for the cases treated with partial adenectomy, 5.0 weeks for those treated with incision and drainage and 6.2 weeks for the abortive forms with spontaneous cure. Up to this time (six months after conclusion of the investigation) none of the 85 surgically treated cases developed chronic edema or elephantiasis of the genitalia, although in 27 per cent bilateral lesions were present. From these results we may conclude that surgical intervention in acute lymphogranuloma inguinale can be regarded as a successful method of therapy, partial adenectomy resulting in earlier healing of the suppurating bubo than does simple incision and drainage.

TREATMENT OF CHRONIC LYMPHOGRANULOMA INGUINALE

While our various therapeutic attempts in the acute lesions of lymphogranuloma inguinale at least can claim some degree of success, we have to admit that treatment of the late or chronic lesions

of the disease gives most unsatisfactory results. Our modern conception of the evolution of this infectious venereal disease however may explain our failure in the therapy of its chronic manifestations —esthiomene or ulcerative elephantiasis and inflammatory stricture of the rectum. Except in the rare cases where general dissemination of the virus can be successfully established, the infectious agent will chiefly inhabit the regionary lymph glands in the area of the primary infection and will have the tendency, after destruction of the glandular tissue, to spread locally. Through lymphstasis in the tissues whose lymph drainage has been interrupted by the destruction of the glands, a chronic edema will be produced. The inflammatory process spreading retrograde along the lymph vessels and adjacent tissues results in a characteristic granulation tissue production with numerous small abscesses and sinuses, the lesion showing marked tendency to massive hyperplastic fibrosis. The development of this chronic manifestation will depend largely upon the location of the affected lymph glands which correctly must be regarded as the center of the entire pathologic process. If the inguinal, presymphyseal or crural lymph nodes are involved, the external genitalia (scrotum, penis and vulva) will bear the brunt of the spreading infection, and elephantiasis of the affected part will develop. An infection of the anorectal glands, the deep iliac and sacral glands will cause a disease of the perineum and rectum—the genito-anorectal syndrome or lymphogranuloma inguinale of the perineum and inflammatory stricture of the rectum. The skin and mucous membranes, however, covering the edematous tissues will suffer considerably from nutritive disturbances caused by the chronic lymphedema and will allow penetration of secondary invaders into the subepithelial structures. Thus severe infections may be established in the already altered tissues. This will hasten their complete destruction. This secondary infection of tissues infected for a long time with the virus of lymphogranuloma inguinale is an important factor in the complications, producing severe ulceration in the instances of elephantiasis—described as esthiomene—and severe proctitis in the cases of rectal stricture.

From this brief sketch of the development of the chronic lesions of lymphogranuloma inguinale we can realize how futile any therapy will be in advanced cases which represent merely an end result of the infection complicated by various secondary pyogenic processes. The only relief the patient can expect is cure of the ulcers in the colon and on the genitalia. The truth of this statement is well borne out by our practical experience in the treatment of esthiomene and rectal stricture. Surgical excision of the parts suffering from elephantiasis is the ideal way of treating esthiomene

but it is only possible in cases where small areas of the genitalia are involved. Antisyphilitic therapy and the injection of tartar emetic have met more with failure than with success and cannot be recommended. Specific treatment with vaccines or Frei antigen has not been studied sufficiently to permit any conclusion but in the few cases used has not given very encouraging results. In the treatment of rectal stricture we are also lacking any specific or even effective therapy. According to Martin the disease in this stage may be described as incurable, tending always towards an inevitable fatal termination. Drastic resection of the rectum, sigmoid, the perirectal tissue and the perineum has brought permanent relief but represents a rather drastic procedure[15]. Simple extirpation of the rectum does not remove the principal site of the pathologic process and therefore recurrence will be observed in a large number of the cases so treated[3]. Colostomy is often a necessary emergency procedure and in most cases the opening must remain permanent. It carries a mortality of about 30 per cent. Gohrbrandt[9] suggests solganol in early cases of rectal stricture and Gay-Prieto[8] observed one case where distinct improvement was noted after intravenous therapy with Frei antigen. A similar observation has been made by Alley[1] who treated nine cases of mild stricture of the rectum with intradermal injections of antigen and obtained "encouraging results." Repeated dilatation of the stricture is the adopted procedure in all not too far advanced cases observed in the Charity Hospital and has met with fair success insofar as delaying the more serious symptoms which, as a rule, require colostomy. Unfortunately, the carelessness of the patient, who so often postpones regular treatment as soon as slight improvement is noted, hinders considerably successful application of this method of therapy. The heavy burden placed upon charity institutions such as ours by attempting to treat rectal stricture by continuous dilatation can be approximately estimated from the short analysis of all cases that received hospital treatment for this purpose during the years 1934-35. The total number of ambulatory cases who similarly received clinic treatment cannot be estimated but is incomparably higher as only the severe cases were admitted to the wards of the hospital.

TABLE II

HOSPITAL TREATMENT OF RECTAL STRICTURE

Total number of cases	*Number of cases admitted for:* 1	2	3	4	5	6	7	8	*longer*	*Readmissions*	*Deaths*
					weeks						
1934											
120 cases	35	28	32	7	6	1	3	4	4	25	7
1935											
116 cases	29	31	24	4	7	3	5	2	9	27	4

A total number of 236 cases of rectal stricture attributable to chronic lymphogranuloma inguinale were admitted during those two years with over 75 per cent of the admissions lasting one week or longer (up to 22 weeks). About 25 per cent of the cases were readmissions for repetition of treatment, in some instances the patient having been admitted four to five times during a year. In the series, eleven deaths occurred, most of them caused by spontaneous or postoperative peritonitis. In two cases dilatation caused rupture of the rectum; in four cases peritonitis followed colostomy.

We believe we have sufficiently stressed in this brief paper the complete failure of therapy in chronic lymphogranuloma inguinale and the question may arise if the future will bring more successful methods of treatment. Considering the pathologic changes which form the basis of the chronic manifestations of the disease, we are not sanguine as to the possibilities of specific therapy though an attenuated virus is hoped for by some authors. The destruction of the tissues and the replacement fibrosis is usually far advanced when the patient forced by the ulcerations or secondary proctitis seeks medical aid and we then no longer deal with a specific progressive disease which could possibly be arrested by specific therapeutic measures but with a permanent tissue defect which no therapy can cure. Therefore our attention should be directed most intensively towards the prevention of this incurable stage of lymphogranuloma inguinale through proper therapeusis of its acute stage. In the female this stage is frequently veiled by the deep location of the affected glands which prevents possible effective early treatment and so explains the greater prevalence of chronic, inoperable, incurable lymphogranuloma inguinale lesions in such patients. This opens a new field of investigation to the gynecologist whose attention should be directed towards the recognition and the early therapy of acute lymphogranuloma inguinale.

Summary

On the basis of the etiology and the pathology of lymphogranuloma inguinale, the various methods of treatment are analyzed and discussed. Surgical removal of the bulk of infected glands, which constitute dangerous foci for the further spread of the disease, can be recommended. The curative effects of partial adenectomy is demonstrated by study of a series of cases so treated. The hopelessness of any type of therapy in the chronic forms of the disease, esthiomene and inflammatory stricture of the rectum, are emphasized and explained on the basis of the character of the pathologic lesions encountered in these conditions.

REFERENCES

1. Alley, R. C.: Symposium on Rectal Stricture (lymphopathia venerea involving rectum), Tr. Am. Proct. Soc. *33:* 150, 1934.
   Also in Kentucky M. J. *32:* 250 (May) 1934.
2. D'Aunoy, R.; von Haam, E., and Lichtenstein, L.: The Virus of Lymphogranuloma Inguinale, Am. J. Path. *11:* 737 (Sept.) 1935.
3. Catellier and Weiss: Pathogenie et traitement des rectites proliferantes et stenosantes, J. Chir. *44:* 554 (Oct.) 1934.
4. Chevallier, P., and Bernard, J.: La maladie de Nicolas-Favre: Lymphogranulomatose inguinale subaiguë, Rev. de med., Paris, *47:* 856, 1930.
5. Chevallier, P., and Fiehrer: Sur le traitement de la maladie de Nicolas et Favre par le salicylate de soude, Bull. Soc. franc. de dermat. et de syph. *40:* 1747, 1933.
6. Chevallier, P., and Fiehrer: Sur le traitement de la maladie de Nicolas et Favre par le salicylate de soude, Bull. Soc. franc. de dermat. et de syph. *40:* 1502, 1933.
7. Coutts, W. E.: Contribution to the Knowledge of Lymphogranulomatosis Venerea as a General Disease, J. Trop. Med. & Hyg. *39:* 13, 1936.
8. Gay-Prieto: El tratamiento biologico de la linfogranulomatosis subaguda con inyeciones intravenosas de antigeno specifico, Arch. de Medicina Cirurgia y Especialidades *35:* 985, 1932.
9. Gohrbandt, E.: Miterkrankung des Rektums und des Urogenitalsystems bei Lymphogranulomatosis inguinalis, Arch. f. klin. Chir. *177:* 611-617, 1933. (Review —Deutsch. med. Wchnschr. *98:* 164, 1934.)
10. Von Haam, E., and D'Aunoy, R.: Is Lymphogranuloma Inguinale a Systemic Disease? In Press.
11. Hellerstroem, S.: Therapeutische Umfrage. Welche Behandlungsmethoden haben sich bei der Lymphogranulomatosis inguinalis bewahrt? Sonderabdruck aus Dermatol. med. Wchnschr. *28:* 868, 1935.
12. Hellerstroem, S.: Das Lymphogranuloma inguinale. (Atiologie und Therapie). Sonderabdruck Deutsch. med. Wchnschr. *28:* 1, 1935.
13. Jersild, O., and Kristjansen, A.: Considérations sur l'affet curatif de l'exèrése ganglionnaire partielle dans la lymphogranulomatose inguinale. (Betrachtungen über den Heilungseffekt der teilweisen Lymphdrüsenausräumung bei der Lymphogranulomatosis inguinalis). Deutsch. med. Wchnschr. *98:* 701, 1934. Also in Bull. Soc. franc. de dermat. et syph. (Reunion dermat.) *40:* 1589 (Dec.) 1933.
14. Kalz, F., and Sagher, F.: Zur Therapie des Lymphogranuloma inguinale. Dermat. Wchnschr. *97:* 1754-1761 (Dec. 16) 1933.
15. Lee and Vander Veer: Radical Resection of the Sigmoid Perirectal Tissue and Perineum in Lymphopatia Venerea. Report of the 61st meeting of the Society of Clinical Surgery. Philadelphia: 1935: 5.
16. Lesueur and Florent: Contribution à l'étude de la lymphatexie; les adénites d'apparence palustre, Arch. de méd., Paris *66:* 64, 1896.
17. Loehe: Therapeutische Umfrage: Welche Behandlungsmethoden haben sich bei der Lymphogranulomatosis inguinalis bewährt? Sonderabdruck aus Dermatol. Wchnschr. *28:* 871, 1935.
18. Loehe and Bluemmers: Weitere Mitteilungen ueber die Lymphogranulomatosis Inguinalis, Med. Klinik *27:* 614, 1931.
19. Mamou: Maladie de Nicolas-Favre conjugale. Intolérance au traitement stibié. Bull. Soc. franc. de dermat. et syph. *39:* 708, 1932.
20. Marbury, W. B.: Lymphogranuloma Inguinale, South. Surgeon *4:* 312 (Oct.) 1935.
21. Martin, C. F.: Stricture of the Rectum: Some of its Problems, J. A. M. A. *101:* 1550 (Nov. 11) 1933.
22. Martin and Calatayud: Tratamiento de la linfogranulomatosis inguinal subaguda, Ars Medica *9:* 58, 1933.

23. Nicolas and Favre: Traitement radiothérapique de la lymphogranulomatose inguinale subaigue, Compt. rend. Soc. de biol. *85:* 472, 1921.

24. Ravaut, Levaditi and Maisler: La valeur diagnostique et thérapeutique des injections intraveineuses du virus de la maladie de Nicolas-Favre d'origine simienne, Bull. Soc. franc. de dermat. et syph. *39:* 1262, 1932.

25. Rousseau, G., and Adamesteanu, C.: Considérations sur le traitement chirurgical de la lymphogranulomatose inguinale bénigne (Maladie de Nicolas-Favre), Presse Med. *42:* 1489 (Sept. 22) 1934.

26. Ruge: Therapeutische Umfrage. Welche Behandlungsmethoden haben sich bei der Lymphogranulomatosis inguinalis bewährt? Sonderabdruck aus Dermatol. Wchnschr. *28:* 873, 1935.

27. Serefis: Therapeutische Umfrage. Welche Behandlungsmethoden haben sich bei der Lymphogranulomatosis inguinalis bewährt? Sonderabdruck aus Dermatol. Wchnschr. *28:* 874, 1935.

28. Sezary and Lenegre: Le traitement de la maladie de Nicolas-Favre par l'Antimoine, Bull. Soc. franc. de dermat. et syph. *39:* 1183, 1932.

29. Sorley and Gibson: Observations on the treatment of climatic bubo and allied diseases, Lancet *2:* 1365, 1933.

30. Stannus, H. S.: A Sixth Venereal Disease. Baltimore: William Wood, 1933.

31. Tamura, J. T.: The Treatment of Lymphogranuloma Inguinale with Vaccine and Antiserum, J. Med. *16:* 178 (June) 1935.

32. von Veress, F.: Beiträge zum gehäuften Vorkommen der Lymphogranulomatosis inguinalis und deren Behandlung, Dermat. Wchnschr. *96:* 201-203 (Feb. 11) 1933.

33. Wien, M. S., and Perlstein, M. O.: Intradermal Treatment of Lymphogranuloma Inguinale: Preliminary report, Arch. Dermat. & Syph. *28:* 42-43 (July) 1933.

PERFORATION OF A PRIMARY JEJUNAL ULCER

D. P. HALL, B. S., M. D., F. A. C. S.
Louisville

The occurrence of acute secondary ulcer of the jejunum subsequent to gastro-enterostomy or partial gastrectomy complicated by an acute hemorrhage or perforation is not infrequent. However the occurrence of a primary jejunal ulcer with perforation appears to be a rare lesion. No space is devoted to it in surgical texts and there is only an occasional reference in surgical literature. Richardson in 1922 presented a resume of twelve diagnosed cases of primary jejunal ulcer, perforation occurring in ten cases. Patterson Brown analyzed thirty-five collected cases of primary jejunal ulcer inclusive of the year 1924. I have collected only seven cases from the literature where the ulcer was of a chronic type from which one may infer that most primary jejunal ulcers are of the acute variety compared to the number reported. From a study of the material available it would seem that most ulcers occurring in the jejunum and so diagnosed by operation are of the acute erosive type proceeding to early perforation and bearing no relation to their sister ulcer of the gastro-jejunal stoma, differing radically from duodenal ulcer with its chronicity, fibrous tissue replacement, and demonstrable obstructive symptoms, but presenting a distinct parallelism in its proneness to perforate.

REPORT OF CASE

Miss B. L., aged 38, was admitted to SS. Mary and Elizabeth Hospital at 8:30 a. m., Aug. 24, 1928, with the following history: Three hours earlier she had been awakened with sudden intense, continuous, burning, stabbing pain situated around the umbilical region followed in an hour by a generalized abdominal pain. Severe pain had been complained of near the tip of the right scapula, so-called phrenic pain. Some mucus had been vomited but no blood. Sodium bicarbonate had been administered with no appreciable diminution in pain. On the arrival of her physician she was found with pallor, coldness, subnormal temperature, blood pressure 92/80, pulse 60, thin and wiry. Her attendant administered caffeine sodium benzoate with morphine which was followed by partial relief. Four years previous to this attack she had suffered recurrent attacks of indigestion three hours after the ingestion of food, lasting for an hour. These attacks had not been relieved by any medicament and had persisted for eight weeks. At the time her stools had been examined and occult blood reported. A Wassermann test had proved negative. Roentgen studies had been made of her gastro-intestinal tract but no lesion had been demonstrated. After the administration of the barium meal there had been an abatement of all symptoms and perfect health ensued until her present illness.

From the Louisville Surgical Society.

Physical Examination: The patient appeared ill, her face was drawn; temperature was 99, pulse 68; blood pressure 100/80. No deviation from normal was elicited upon examination of heart and lungs. The abdomen was retracted with limited movement of abdominal muscles. Rigidity and tenderness were generalized. Respirations were jerky and shallow, with difficulty in speech. Hepatic dulness was replaced by a high pitched tympanitic note. Slight shifting flatness was present in the lower abdomen. A catheterized specimen of urine was obtained; specific gravity 1.018, no albumin, no sugar, no pus cells and no red blood cells or casts. Her hemoglobin was 78 per cent; white blood cells 11,200, polymorphonuclears 80 per cent, red blood cells 4,100,000. Blood sedimentation time was 60 minutes. A diagnosis of perforation of a duodenal ulcer was charted.

Operation was performed 4½ hours after onset of symptoms. An upper right paramedian incision was used. The parietal peritoneum was a dusky red. On incising this there was a gush of green watery fluid accompanied by gas, lymph flakes, and small dark particles of food. The gastric, intestinal, and gallbladder serosa were congested. The fluid was evacuated by Poole's suction tip after which the duodenum was carefully inspected for perforation. The stomach was also examined but no visible leak was found. The pylorus was patent, no scars or contractions were noted. In conducting the preceding exploration a large amount of green fluid was seen to well up from under the transverse colon synchronous with expiration. The transverse colon was delivered extraperitoneally exposing a large pool of green fluid limited above by the mesocolon. On displacing the small intestine downward and evacuating the fluid a spurt of green fluid ensued from a perforation on the antemesenteric border of the jejunum, about 3 inches from the ligament of Treitz. The perforation was approximately 1.5 mm. in diameter with a small flake of lymph adherent to the edge. A small area of extreme reddening with slight induration encircled the opening. A cautery tip was introduced destroying the ulcer area. Two Babcock intestinal forceps were applied to make transverse traction. Closure was effected by the application of one row of Czerny Lembert sutures, inverted by mattress sutures of chromic catgut. The ends of the mattress sutures served to fix an omental graft. Very slight diminution in the lumen of the jejunum resulted. One rubber tube drain was introduced into the abdomen through a suprapubic stab wound. Closure was effected in the usual manner. A Levine duodenal tube was introduced nasally and a suction apparatus connected for drainage. Ten per cent glucose in saline was administered by venoclysis for three days. A smooth convalescence devoid of distention and a minimum degree of pain followed. The absence of drainage permitted removal of the suprapubic drain on the fourth postoperative day.

Eight years have elapsed and on inquiry we find she has experienced no subsequent recurrence of symptoms.

In conclusion I am not prepared to state definitely the etiologic factors of primary jejunal ulcer, but I am impressed by the fact that displaced gastric mucosa in a Meckel's diverticulum often produces symptoms and end results similar to the foregoing, as reported by Curd[1]. One might by analogy presuppose the same pathologic process occurring in the jejunum. I would call to your attention in all

cases exhibiting the clinical feature of a gastro-intestinal perforation regardless of a previous negative x-ray series, the possibility of a primary jejunal ulcer complicated by perforation.

REFERENCE

1. Curd, H. H.: A Histologic Study of Meckel's Diverticulum with Special Reference to Heterotropic Tissue, Arch. Surg. *32:* 506-523 (March) 1936.

The Southern Surgeon

Published Bi-monthly by

The SOUTHERN SURGEON PUBLISHING COMPANY

478 Peachtree Street, N. E.

ATLANTA

VOLUME V AUGUST, 1936 NUMBER 4

PRINCIPLES FOR THE TREATMENT OF IMPORTANT FRACTURES

The chief requisites for prompt union may be submitted in the order of their importance:

Regional nutrition.

Alignment of the fragments.

Approximation of the fragments.

When it is remembered that bone receives its principal nutrition from the circulation in the soft tissues by which it is surrounded, the importance of avoiding embarrassment of the capillaries in the adjacent tissues is readily appreciated. Any external means of immobilization however skillfully and carefully employed is sure to cause some interference with the circulation in the part involved and this I regard as the chief cause of non-union. If it were possible to treat fractures of the long bone without the encumbrances of external immobilizing apparatus, non-union of bone would be as rare as non-union of skin or fascia.

The prejudice existing today against direct fixation of broken bones is based upon the fear of infection in open operation. When some form of confining apparatus is immediately applied which compromises the circulation to the extent that the wounded tissues are inadequately supplied with nutrition, these tissues are not able to offer normal resistance against the invading organisms.

When I was a hospital intern the dictum was handed down to apply plaster spicas on all cases of inguinal hernia that were operated upon. The result was that in at least three-fourths of cases there was suppuration and the practice was discontinued. For the same reason open wounds that involve fractures of long bones yield to bacterial contamination in the same manner.

It has been found in practice that fragments of long bones which are placed in alignment and maintained in position will unite in spite of considerable lack of contact in their proximal and distal extremities but if alignment is imperfect good union is extremely difficult to obtain.

We have found that the period required for bony union is reduced to one-half the time ordinarily required if the fragments are maintained in alignment by internal fixation alone and that infection following open operation with rigid asepsis is a rare complication. One advantage of direct fixation is that splinters and small fragments can be removed and sharp ends cut away until smooth surfaces of both fragments are left for approximation.

The materials for obtaining fixation are quite numerous and offer a long list to select from. They can be divided into metallic and osseous substances. In the metallic group plates, wires, bands and nails head the list. I have always felt that these metal substances are more irritating to the bone tissues than substances of the same character and origin as the bone itself. Autogenous graft are probably the most popular but they can only rarely be obtained in sufficient size to maintain the proper alignment without the use of external measures. Their procurement exposes the patient to as great a hazard as the operation for uniting the fragments. We have found in practice that beef bone if properly prepared, of sufficient size and proper shape meets all the requirements in a most satisfactory manner. It works particularly well in compound fractures, gunshot fractures, non-union, and even in the presence of infection. In every instance it seems to fertilize the fracture and promotes the growth of new bone. I have been astonished to see callus formation begin in a case of non-union of a femur that had been ununited over two years and complete union occur in four weeks. In forty-two cases involving the femur in which I have used the beef bone key most of them had been submitted to every other known means of treatment without success.

Fractures involving the upper end of the femur in old people present the simplest and easiest requirements we have to deal with. This includes fractures of the neck (intracapsular), intertrochanteric and subtrochanteric.

The bone key which we use is driven through the cancellous bone at right angles to the line of fracture, no holes are drilled and one is guided by the x-ray film. Before the operation is begun the two limbs are posed in symmetrical positions. The whole procedure can be completed in ten to fifteen minutes. No form of external immobilization is employed and the patient is permitted to assume any position he chooses after he is placed in bed.

In a group of eighty-one patients ranging in ages from 62 to 86 years there has been no mortality except from collateral disease that was pre-existing at the time of injury. In no case was there a failure of union or confinement of more than four weeks.

Our contentions are that bony regeneration is both rapid and ample if local nutrition is not embarrassed or impeded and that under the same circumstances bone will resist the invasion of bacteria with the same vigor and success that fascia does. That metallic substances placed in contact with bone fragments possess greater aptitude for insult than material of the same nature as above. We see an illustration in the absence of visible reaction of skin when sutured with horse hair, both being epithelial structures.

The mechanics of the plan have been described in detail and published in various journals and it would be superfluous to include them here.

G. A. HENDON, M. D.

CONQUEST OF PAIN

C. C. Howard, M. D.
Glasgow, Ky.

God made Adam and placed him in the Garden of Eden to enjoy this grandeur of nature forever. Here, in this open paradise where sin was unknown, God caused Adam to fall into a deep sleep and removed his rib without pain. This is the first operation and the first anesthetic.

The First Operation Performed Under Ether
From the original oil painting by Maurice Siegler

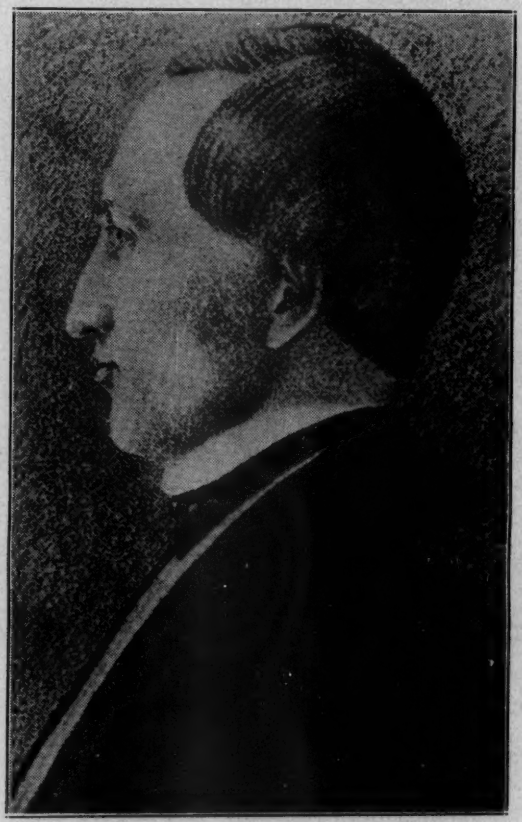
The second phase of this great adventure is the tragedy of all the ages. Adam and Eve partook of the forbidden fruit, and then sin and pain entered into the world. Let us stop and think of the countless millions who have suffered physical and mental pain since that fatal day! Briefly, I shall attempt to trace the history from that date to the present generation.

Among the many crude ways to relieve pain during the period before Christ, only one stands out as a real contribution, opium.

Read before the Seventh Annual Assembly of The Southeastern Surgical Congress in New Orleans, March 9, 10 and 11, 1936.

We will never know how some unknown person from Asia Minor happened to cut the poppy capsule and taste the juice, thereby learning it would give relief from pain. This unknown person is the true pioneer of the ages; his monument should stand by Pasteur's as a benefactor to mankind.

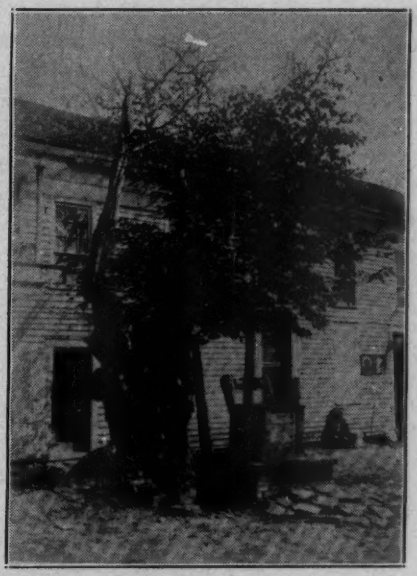
Asia Minor is the home of the poppy. There, the climate, soil and people are blended together in just the right proportion to make



CRAWFORD W. LONG, aged 26

the highest grade opium in the world (it contains 10 per cent morphine). The opium of India and China has a low grade morphine content and is used for smoking.

Opium was used in a crude way until 1669, when Thomas Sydenham of England extracted the tincture of opium, which was the sheet



Stairway entering the building in which two operations on Venable were performed. To the left on the ground floor was Dr. Long's office, in the rear his drug shop.

anchor for almost two centuries. In 1815, Ser Turner of Germany extracted from crude opium a substance which was alkaline in reaction (the first alkaloid). This he named morphine after the god of sleep.

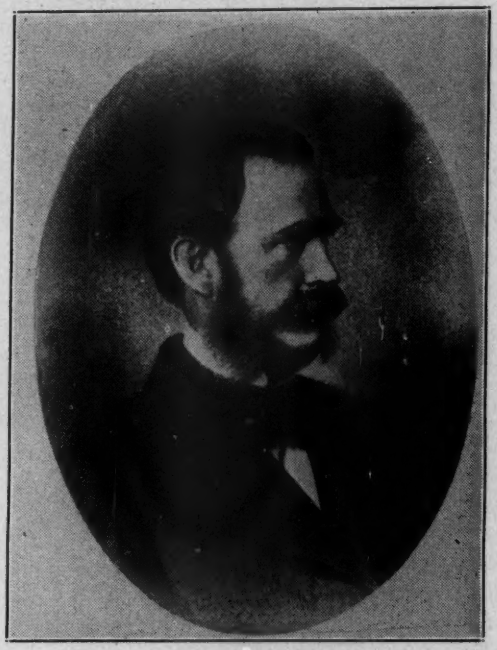
Valerus Cordus, German chemist, in the year 1535, was the first to make ether by distilling a mixture of sulphuric acid and alcohol. It was three hundred years later that its great usefulness was found through the genius of two young men of a new continent.

One of the great discoveries in the history of the world was made by Crawford W. Long, a young country doctor of Jefferson, Georgia.

Dr. Long attended medical lectures at old Transylvania, later graduating from the Medical Department of the University of Pennsylvania. Early in his college life he was a roommate of Alexander H. Stephens, later Vice-President of the Confederacy. These roommates were destined to represent Georgia in the Hall of Fame.

Having noted that young men on an "ether frolic" experienced bruises without pain, Dr. Long reasoned that this phenomenon might be utilized in surgery. At the age of 26 therefore he adminis-

tered ether drop by drop on a towel until his patient, young Venable, was asleep. (This method of administering ether has not been improved upon.) Then without causing pain, Dr. Long removed a tumor from his neck. This is the first operation performed by man without pain. There in a primitive office in a backwoods town, we have the discoverer of anesthesia in 1842.



W. T. G. MORTON, 1819-1868
From the "Semi-Centennial of Anesthesia," Boston, 1896.

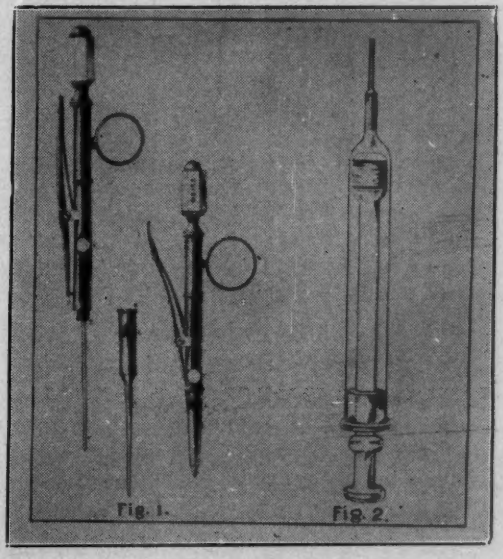
After his successful experience with ether in the case of young Venable, Dr. Long used it in obstetrics, for amputations and for removal of breasts. He proved its value beyond the peradventure of a doubt. He was the genius who unlocked the door that led to the modern hospital; he lit the candle of anesthesia which has burned brighter and brighter during these hundred years.

At the ripe age of 74, while at the bedside of a patient in confinement, Dr. Long suddenly slumped over and his heroic soul floated away to immortality.

Four years after Long had first used ether in a surgical operation, W. G. T. Morton, a dentist of Boston then studying medicine at Harvard, through reasoning similar to that of Long's, experimented

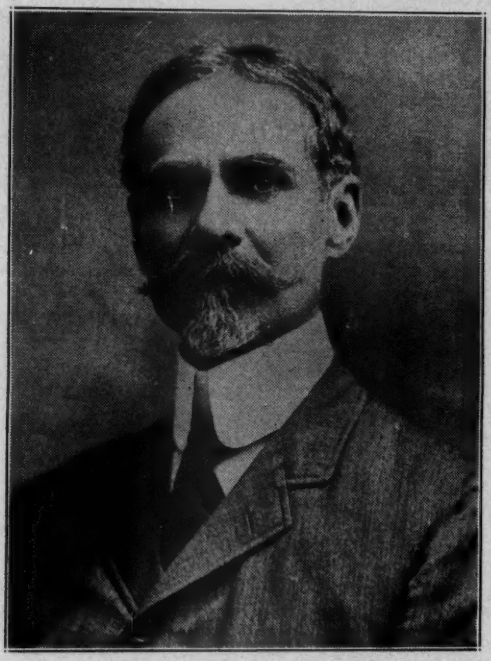
Dr. Morton demonstrating the administration of ether at the Massachusetts General Hospital, October 16, 1846.
From the "Semi-Centennial of Anesthesia," Boston, 1896

with ether to relieve the pain of dentistry. It gave the desired results in dentistry so he asked Dr. Warren, chief of the surgical staff of the Massachusetts General Hospital, for permission to demonstrate it. Dr. Warren appointed the amphitheater of the hospital on October 16, 1846 for the experiment.



The First Hypodermic Syringe

Dr. Morton appeared at the hospital a few minutes late. He had with him a new mask for administering the drug. In a short time the patient was ready. Dr. Warren proceeded to remove a tumor from under the lower jaw, and immediately announced to the audience, "Gentlemen, this is no fake." That was the first public demonstration of painless surgery. Oliver Wendell Holmes suggested the word anesthesia.



LEONARD CORNING, M. D., 1855-1923

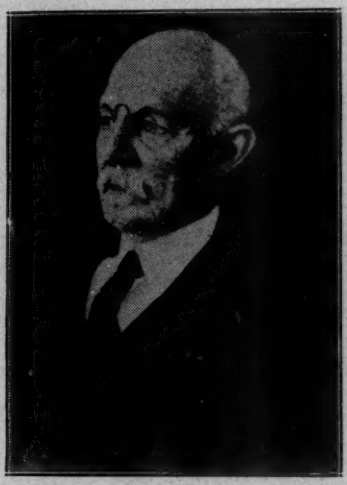
The next step up the ladder was the crude hypodermic syringe devised by Doctor Rynd, of Dublin (1845).

In 1846, Sir James Y. Simpson, of Edinburgh, introduced chloroform into the field of general anesthesia.

An English lady born in Florence, Florence Nightingale early in life began ministering to man and beast. Her great opportunity came during the Crimean War. English soldiers were dying like sheep on the shores of the Black Sea without nursing or well organized medical service. Miss Nightingale secured permission from her government to go to Scutari to care for the sick and dying. Her work there with the British Tommies raised her to the Saint-

hood of Nursing and there on Turkish soil was the beginning of our modern nursing.

At the end of this period we had passed through the twilight zone of medicine. The discoveries and developments in the twelve years



WILLIAM STEWART HALSTED, M. D., 1852-1922

CARL KOLLER, M. D.

between 1841 and 1854 gave to civilization more happiness and joy than all the others since the birth of Christ. Enumerating them we have,

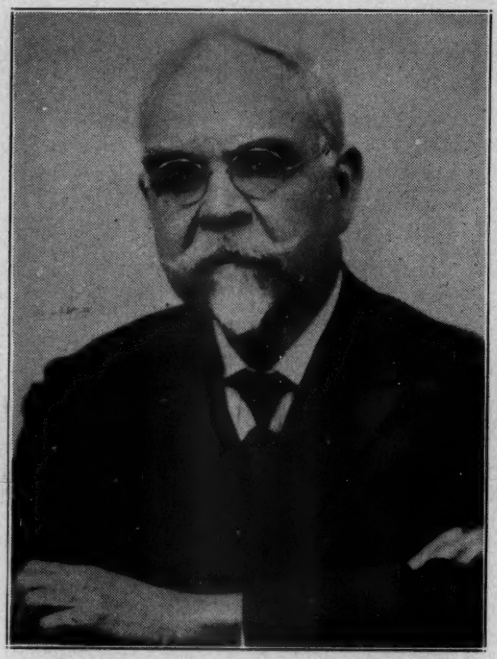
1. Ether, 1842.
2. Hypodermic syringe, 1845.
3. Chloroform, 1846.
4. Professional nursing, 1853.

These four have given to the world the greatest instruments that have ever been placed in the hands of our profession. How little and how helpless we would be without them!

In the year 1844 Horace Wells, a former partner of Morton's, used nitrous oxide as an anesthetic. During a period of heated controversy as to who was the real discoverer of anesthesia, Wells committed suicide. Through it all Crawford W. Long continued the even tenor of rural practice in Georgia.

The cultivation and chewing of the coca plant is older than our history. The native Indian was using it when Pizarro conquered Peru. They called it "the divine plant." The leaves are dried in the

sun, then packed for sale. The natives chew the leaves after mixing them with a little lime. This is said to give endurance. In 1860 Newman extracted cocaine from the coca leaf. It is a long way from



DR. RUDOLPH MATAS

the Indian on the slopes of the Andes with his quid of coca leaves and lime to the use of cocaine in modern medicine.

Cocaine was first proposed as a local anesthetic by Carl Koller in 1884. He was then house surgeon in the Allgemeine Krankenhaus in Vienna. While experimenting with cocaine as a substitute for morphine in the treatment of addicts, Koller noted the peculiar effect of cocaine on the tongue when taken orally. A report of that characteristic effect had appeared previously in the literature, but had been lost sight of. About the same time he dropped a solution of cocaine in his eye and found that it gave perfect anesthesia. Experimentation in Strickler's laboratory confirmed this and a preliminary communication was presented to the German Ophthalmological Society on September 15, 1884. With this a new and most powerful agent was placed in the hands of man, and experiments in

local, regional and spinal anesthesia were undertaken all over the world.

Following Koller, William Stewart Halsted while working in the Bellevue Hospital of New York, developed regional anesthesia. He later became one of the "Big Four" of Johns Hopkins.

To Dr. Leonard Corning of New York goes the credit for introducing spinal anesthesia. He used 1 per cent cocaine. Dr. Corning proved its possibility, but it took years of painstaking work to develop a high degree of efficiency.

To our own Dr. Rudolph Matas goes the honor of the first public demonstration of spinal anesthesia in America. This operation, a hemorrhoidectomy on a negro boy, was performed in the old Charity Hospital in New Orleans.

Procaine was introduced in 1905. Spinal anesthesia and local anesthesia were then lifted to their present place in the surgical world.

When you think back to the Garden of Eden and the long rough highway of life, mark indelibly into your memory these few heroic souls who have made it possible for you to practice the greatest profession in the world. Let us not forget our great mission: To cure if possible; to relieve pain, but always to spread hope and good cheer.

EXECUTIVE COUNCIL MEETS

The Executive Council met in Atlanta the evening of June 18. Dr. John Darrington, of Yazoo City, elected Vice President at the March Assembly, succeeded to the Presidency. Dr. J. S. Turberville, of Century, Florida, was made Vice President.

A motion was introduced to establish the C. Jeff Miller Memorial Lectureship. Further details of this must be announced later.

The Council attended to numerous details and approved the preliminary arrangements for the Assembly in Louisville next March. It also approved the negotiations under progress for weeks between the Editors of THE SOUTHERN SURGEON and the Louisville Surgical Society by which the Louisville Surgical Society adopts this Journal as its official organ. It also authorized the Editors to make similar arrangements with other similar surgical organizations in the South. Finally, the Council raised the subscription price of the Surgeon to $4.00 a year.

The Atlanta Fellows gave a dinner at the Biltmore in honor of the visitors. Later the Fulton County Medical Society turned its regular meeting over to the Congress, and Dr. Darrington and Dr. Holden addressed the Society.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

PASSIVE VASCULAR EXERCISES: *The Conservative Management of Obliterative Arterial Diseases of the Extremities.* By LOUIS G. HERRMANN, A.B., M.D., Assistant Professor of Surgery, College of Medicine, University of Cincinnati and the Cincinnati General Hospital; Director of the Vascular Disease Clinics of the Cincinnati General Hospital and the Christian B. Holmes Hospital of the University of Cincinnati. With a foreword by MONT R. REID, M D. 288 pages with 80 engravings and 4 colored plates. Price, $6. Philadelphia and London: J. B. Lippincott Company, 1936.

One of the truly great recent advances in surgery has been the rediscovery of alterations in pressure as a means of promoting circulation in an extremity whose principal artery has been occluded.

A fortuitous incident while skiiing in the Alps aroused Dr. Herrmann's interest in negative pressure as a means of promoting circulation in the extremities. He tackled this problem with youthful enthusiasm and sustained assiduity. After making considerable headway and obtaining much clinical experience, careful research revealed that a forgotten Englishman had had the same idea in 1803 and that a number of men since that time had made apparatus for the purpose of producing negative pressure in an extremity. Partly on account of a lack of understanding of physiology, partly on account of lack of care and preliminary training on the part of the operators, such apparatus were not widely used. Perhaps too, though the author does not say so, these contraptions were clumsy and hard to work. However, the field had lain fallow for more than ten years when Dr. Herrmann struck, and when he struck he struck deep.

In the present volume he not only takes up the history of the movement before his day but traces out the development of the technic in his own hands and the results are startling. He emphasizes throughout that careful clinical study and scrupulous attention to detail are of fundamental importance and he intimates that the use of his apparatus by untrained and unskilled men is apt to bring it into disrepute.

Passive vascular exercise is here to stay, and Herrmann has rendered a real service in presenting a full, up-to-date discussion of the whole subject in this monograph.

THE COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION, Volume XXVII (Papers of 1935). Edited by RICHARD M. HEWITT, M.A., M.D., LLOYD G. POTTER and A. B. NEVLING, M.D. 1353 pages with 256 illustrations. Price, $12. Philadelphia and London: W. B. Saunders Company, 1936.

It is extraordinary how The Mayo Clinic manages to keep such a high standard of excellence in its yearly volume. Perhaps one important factor is that in the finished work, of 693 articles published in 1935 from this famous

institution, only 83 appear in full and 458 are listed merely by title. The balance are abridged or abstracted. The book then, represents the cream of the crop, and it is truly cream.

The esophagus accounts for five papers. Esophagitis is rather a new clinical entity. Considerable space is devoted to the physiology of the stomach. One of Dr. Judd's last papers deals with the surgical treatment of lesions of the stomach and duodenum. One notes that more emphasis is laid on the medical management of duodenal ulcer than formerly. One also notes that transfusion seems to have displaced the use of calcium chloride intravenously in jaundiced patients. Twenty-year cures of carcinoma of the colon are inspiring. The dysenteries are well discussed in some practical papers. Alvarez is represented by "Hints for Recognizing the Patient Who will Probably not be Helped by an Abdominal Operation," which appeared in these pages last year, "Unrecognized Strokes and the Gastro-enterologist," "Foods that Commonly Disagree with People" and others. The genito-urinary section is full and has particularly good papers on stones and tumors, as well as Dr. Cabot's "The Management of the Incompletely Descended Testis," which appeared in the SURGEON last October. Fever therapy for gonococcic infections is stimulating.

Dr. Charles H. Mayo reviews 37,228 cases of goiter operated on at the clinic; and not one of these glands was stolen. Hyperparathyroidism and diabetic coma are well discussed. It is interesting to note that Judd felt that "spontaneous hypoglycemia" was to be preferred to Seale Harris' term "hyperinsulinism." This is another rather new syndrome. Adrenal cortex extract is described by Kendall. The Mayos themselves have reviewed more than 1,300 splenectomies and Willius has two splendid practical papers on heart disease. One notes that enthusiasm for Herrmann's apparatus for passive vascular exercise is distinctly limited. O'Leary contributes two authoritative papers on syphilis. New and Figi emphasize that the treatment of carcinoma of the larynx should be individualized, the proper procedure being selected for each case.

Harrington reviews 3,740 cases of carcinoma of the breast. The younger patients were in greater risk of an early death, but nearly half of those under thirty lived ten years or longer. The most interesting paper in the section on neurology is Adson's contribution with regard to the surgical treatment of hypertension, rhizotomy with partial adrenalectomy. The reviewer is not altogether sold on this particular subject.

On the whole, this Mayo volume, like its predecessors, contains much to benefit the general surgeon, the various specialists and the practitioner and will help each to keep abreast of the times.

NEUROLOGICAL SURGERY. By LOYAL DAVIS, M.S., M.D., Ph.D., D.Sc. (Hon.), Professor of Surgery and Chairman of the Division of Surgery, Northwestern University Medical School, Chicago. 429 pages with 172 engravings and 2 plates. Price, $6. Philadelphia: Lea & Febiger, 1936.

When the reviewer picked up this small volume he was surprised that any one should attempt to cover the broad field of neurosurgery in such limited compass. He opened the book without much enthusiasm but then he read from the preface:

"Since 1900, progress in neurological surgery has been so rapid that there is a large number of physicians who are not entirely familiar with what can be accomplished in this field of surgery. There are many excellent and comprehensive monographs which deal with one specific problem of this interesting subject but unfortunately those doctors to whom they would be of most service do not have the opportunity of consulting these authoritative sources of information. As a result, though the diagnosis has been made accurately, one frequently encounters patients who have been given erroneous advice as to just what can be accomplished in the surgical treatment of their condition.

"This book makes no pretense at including an exhaustive treatise upon each subject considered and therefore will be of no help to the experienced neurologist or neurological surgeon. Neither has an attempt been made to give detailed instructions as to the technique of operations in a field which in our opinion requires many years of basic training. The purpose has been to give to the practitioner of medicine easily assimilable facts which will aid him in giving his patient accurate and sound advice."

THE SOUTHERN SURGEON from the beginning has been deeply interested in neurosurgery: this number carries the third of a series of four papers on the subject. Though it agrees with Dr. Davis that the preparation for doing good work in this field must be long and arduous, it believes that the profession should be constantly reminded that surgery of the central nervous system is a rapidly expanding field and such surgery is required in a much larger percentage of cases than was dreamed of thirty years ago. In passing, the SURGEON is proud to note that now there are splendidly trained young neurosurgeons strategically located in the South.

This journal extends to Dr. Davis congratulations on turning out such a valuable piece of work and it earnestly hopes that the book will have as wide a circulation as it deserves.

DISEASES OF THE RESPIRATORY TRACT. Clinical Lectures of the Eighth Annual Graduate Fortnight of the New York Academy of Medicine. By 21 contributors. 418 pages with 56 illustrations. Price, $5.50. Philadelphia and London: W. B. Saunders Company, 1936.

This book is a splendid refresher course in diseases of the respiratory tract. Much of it, it is true, deals with purely medical problems, but fortunately in many respects the sharp line dividing medicine and surgery is fading out. Diseases of the larynx, trachea and main bronchi, bronchoscopy, bronchiectasis, surgery of tuberculosis of the chest, non-tuberculous empyema, abscess and gangrene of the lungs, pulmonary thrombosis and embolism, atelectasis, massive collapse and related postoperative conditions, and carcinoma of the lung are authoritative and well presented. These chapters impress upon one that the surgeon must consider the respiratory tract. They should be studied by the internist too in order that he may know what aid he can expect from the surgeon of today.

Martland contributes a superb section on diseases of the mediastinum. His extensive first-hand knowledge of pathology entitles him to speak with authority and yet he writes with the freshness of a sophomore. It is both entertaining and stimulating to see how he challenges many ideas which have been

blindly accepted from the past. He has little patience with the grading of malignancy. Every physician should be able to derive some benefit from this "somewhat critical review."

EMERGENCY SURGERY. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital; Surgeon and Urologist, Essex County Council, Consulting Surgeon, Clacton Hospital; etc. Second Edition. 842 pages with 812 illustrations. Price, $14. Baltimore: William Wood and Company, 1936.

Mr. Bailey says that it has been his aim "to provide a manual to which the surgical practitioner can turn when he himself must deal with an acute emergency." He has achieved this aim. "When to operate and when not to operate and how to operate under emergency conditions is the theme."

This book is really beautiful. The numerous illustrations in full color add immensely to its value: it is indeed one of the best illustrated medical textbooks that has come our way in some time. The style is charming. The book is full of the common sense and clinical judgment that have over the centuries characterized our profession in Great Britain. It enables one to feel that with the exercise of these qualities a surgeon often can do justice by his patients without expensive and time-consuming procedures; moreover, not infrequently he should operate without such delays.

To be worth his salt a reviewer is supposed to find some imperfections. The only sin of omission that has been noted in this book is that passive vascular exercise in the treatment of arterial occlusion is not mentioned, and the only sin of commission is that certain procedures are included which could only by great stretch of the imagination be considered emergency operations.

The book is enthusiastically recommended, especially to the surgeon who practices in small communities.

MINOR SURGERY. By FREDERICK CHRISTOPHER, S.B., M.D., F.A.C.S. Associate Professor of Surgery at the Northwestern University Medical School, Chicago. Chief Surgeon at the Evanston (Ill.) Hospital. With a foreword by ALLEN B. KANAVEL, M.D., F.A.C.S., Professor of Surgery at the Northwestern University Medical School. Third Edition. Reset. 1030 pages with 709 illustrations. Price, $10. Philadelphia and London: W. B. Saunders Company, 1936.

It is a pleasure to welcome the third edition of this classic. Since, as the author rightly maintains, the larger part of surgery does fall in this classification, this book contains clear instructions for the performance of a large majority of all operations. He also emphasizes that proper minor surgery will often make major surgery unnecessary later. This book should prove helpful to most doctors, for nearly all of us have to do some minor surgery at times. It should prove invaluable to interns.

"Included in the new material are sections dealing with new methods for promoting wound healing, bacteriophage, resuscitation upon the operating table, de Takats method of ambulatory vein ligation, effort thrombosis, leech

treatment of phlebitis, x-ray treatment of gas gangrene, bismuth injection treatment of warts, sodium morrhuate in cystic hygroma, 'sprinter's fracture,' lymphogranuloma inguinale, Elliott treatment of pelvic inflammatory disease, torus fractures, glomus tumors, iliopectineal bursitis, Leriche treatment of sprains, aspiration biopsy, Wangensteen stomach suction apparatus, etc."

It is most interesting to note that a chapter is devoted to the injection treatment of hernia. THE SOUTHERN SURGEON is quite proud of space it has devoted to this newly developed method of treatment, which in the very recent past was frowned upon to say the least.

The last chapter, "The Surgical Intern," should be almost memorized by interns. And while we are at it, the last page or two on the duties of the visiting man to his intern should also be carefully studied by the successful surgeon.

SUBSCRIPTION BLANK

THE SOUTHERN SURGEON is a surgical journal devoted to the progress of surgery, and the only journal of its kind published in the South. Issued bi-monthly.

DEPARTMENTS

GENERAL SURGERY	NEUROSURGERY
UROLOGIC SURGERY	OPHTHALMOLOGY
ORTHOPEDIC SURGERY	INDUSTRIAL SURGERY
OBSTETRICS AND GYNECOLOGY	OTO-RHINO-LARYNGOLOGY

Annual subscription $4.00 in U. S. and Canada. $5.00 foreign. Checks should be made payable to Dr. B. T. Beasley, Managing Editor, 478 Peachtree St., Atlanta, Ga.

Please enter my subscription for THE SOUTHERN SURGION.

_____

_____